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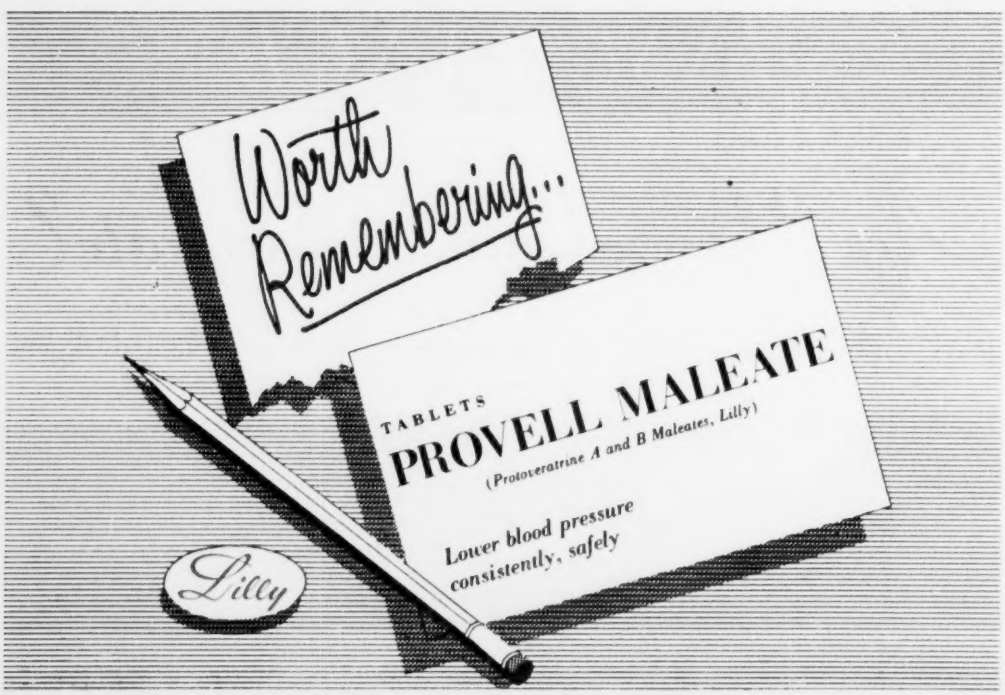

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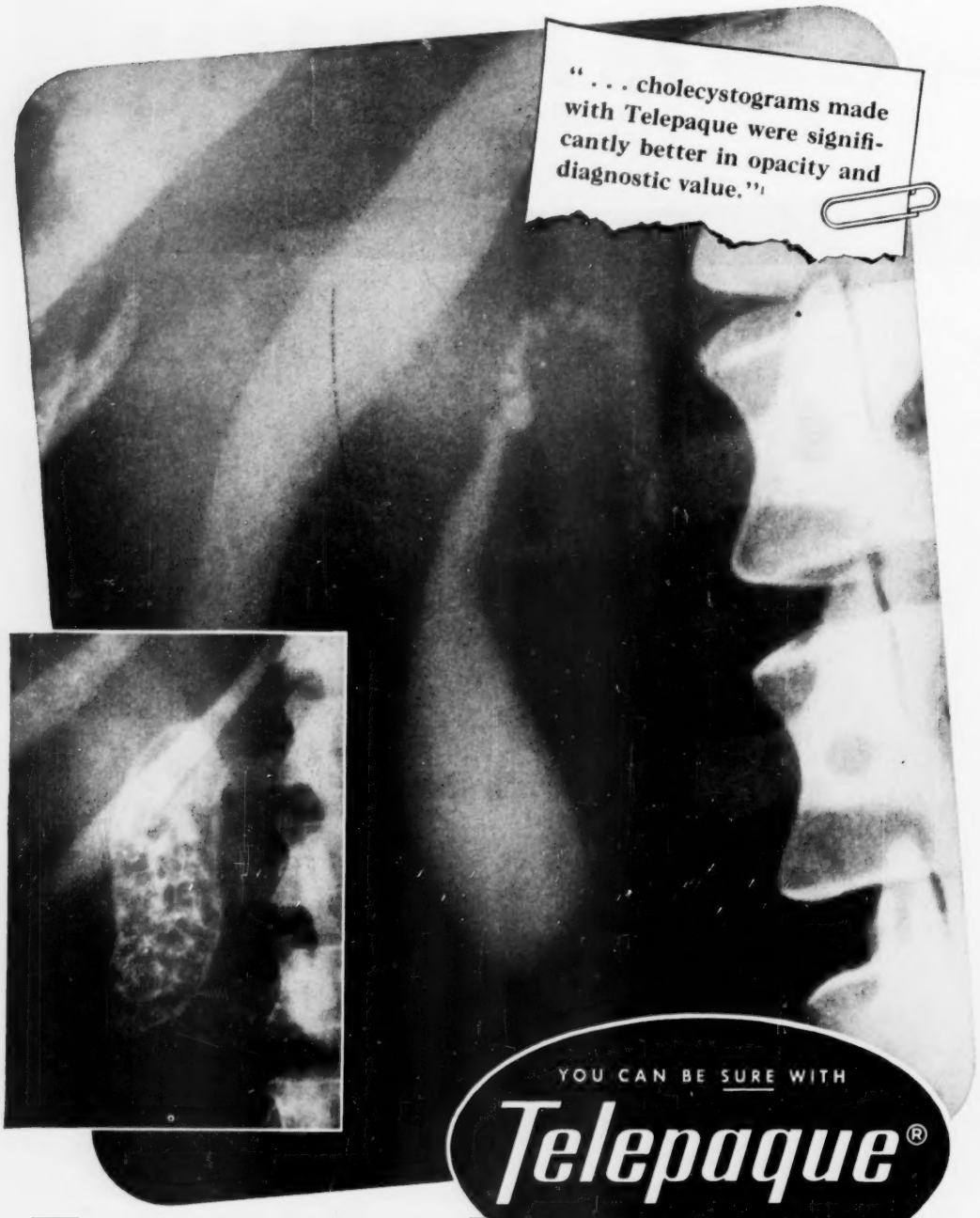
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VAGOTOMY FOR DUODENAL ULCER*

A Final Survey after Ten Years

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BOSTON

IF one could accept completely the time-honored physiologic concepts of gastric secretion and the equally respected "acid-pepsin" theory of the etiology of peptic ulcer, one would be in a position to eradicate duodenal ulcer by surgical measures. One would control the cephalic phase of secretion very neatly by severing the vagus nerves and manage the digestive or humoral phase of secretion by removing the antrum and pyloric ring. The intestinal phase and any other gastric-stimulating factor would then be controlled by removal of the body and fundus of the stomach, and the upper small bowel. Possibly, adrenalectomy would be advisable for the few unfortunate patients whose steroidal pathways were especially active.

This carries physiologic concepts to absurdity. It leaves the hypothetical patient in an even less enviable position than he was with his ulcer. The treatment would be worse than the disease, and he would be a nutritional and economic cripple. The evident absurdity points up two facts of great importance in ulcer disease: that clinical results, not beauty of rationale, must govern the choice of surgical treatment, and that the search for a nonsecreting upper gastrointestinal tract may be clinically unrewarding.

When the evolution of surgery for peptic ulcer is traced, it is clear that surgeons for fifty years have sought an operation that would be simple to accomplish and would still prevent recurrent ulcer. The early procedures such as pyloroplasty and posterior gastroenterostomy were designed primarily as rerouting procedures to overcome obstruction. Posterior gastroenterostomy, while bathing the upper jejunum in the gastric contents, allowed alkaline duodenal contents to neutralize the acid peptic secretions at the stoma. These operations, although still useful at times, did not prevent recurrence in many patients.

With the advent of subtotal gastrectomy, a definitive attack on the acid-pepsin aspect of ulcer disease was envisaged. The early Bilroth I procedure had as its effect the removal of the antrum and at least some of the acid-bearing portion of the stomach. But it was not enough, and recurrences soon appeared. Subtotal gastrectomy (two thirds of the stomach and all of the antrum) combined with gastroenterostomy seemed to be well conceived. The mortality, initially high, was reduced as surgeons became more adept and as supportive measures and anesthesia improved. The incidence of marginal ulcer, also high in the beginning, became lower (3 or 4 per cent) as surgeons performed more adequate gastrectomies and paid attention to the antrum and pyloric ring. But there were still problems related to subtotal gastrectomy that stimulated an interest in improving the surgery of peptic ulcer.

The result of this interest was vagotomy. In the ten years since its introduction on a large scale, certain definite points have been clarified. Many of these aspects have been brought out by a report of the Subcommittee on Vagotomy of the Committee on Peptic Ulcer.[1] Others have been substantiated by reports by Grimson and his associates,[2-4] Crile, Jones and Davis,[5, 6] Dragstedt et al.,[7, 8] Moore,[9, 10] Brooks and his co-workers,[11] Moore and his associates,[12, 12a-12c] Farmer et al.,[13] Stein and Meyer,[14] Colp and his collaborators,[15, 16] Walters, Belding and Lillie[17] and Glenn.[18]

It is clear that the *mortality* after subtotal gastrectomy is consistently higher (3 to 10 per cent) than that after vagotomy (0 to 3 per cent) or vagotomy and posterior gastroenterostomy (2 to 4 per cent).

It is hard to decide which of the *side effects* are worse. The patient with subtotal gastrectomy may suffer from intestinal and nutritional symptoms. The patient with vagotomy complains of heartburn and fullness after eating, noticeable abdominal pain on occasions, and often a very intractable

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form of diarrhea. Many of these by-products are abated by supplementary posterior gastroenterostomy. Despite these discouraging aspects of ulcer surgery, we again emphasize that poor results are in the minority. Both in this group and in a previously reported group of 175 gastrectomies,[12] the vast majority of patients have been well served by their surgeons.

The efficacy of any surgical procedure for duodenal ulcer depends upon the frequency of *recurrence.* Slight differences in mortality or side effects are obviously important factors to consider, but once stabilized as a procedure that can be carried out with a "reasonable mortality and morbidity," any operation for ulcer must stand on its record of recurrences. This is the essence of ulcer surgery, and this paper reviews a late study of vagotomy in an effort to ascertain if empirical results (as measured by recurrence) justify the continued use of this procedure.

A follow-up analysis of the patients who had vagus resection at the Massachusetts General Hospital between 1943 and 1948 is presented. There were 132 patients in all, most of them having vagotomy in 1946 or 1947. There were a few operations in 1943, and some in 1948. The span of the follow-up period is therefore from six to ten years. This was a group of patients from various walks of life chosen for vagotomy because certain aspects of their disease seemed to make them good candidates. Young patients were sought, particularly males, with high levels of emotional response to their work or surroundings because of the presumed central-nervous-system aspect of their disease. Patients with severe bleeding or obstruction were not considered. The majority had "pure" transthoracic vagotomy because of the firm conviction that only with a normal, intact stomach could the physiologic results of vagotomy be ascertained clinically and studied scientifically.

There was also a group of patients who had had posterior gastroenterostomy and entered the hospital with marginal ulcers. In a few patients gastroenterostomy had been performed at the time of abdominal vagotomy; in a few others vagotomy for marginal ulcer had followed subtotal gastrectomy; finally, there were a few in whom posterior gastroenterostomy was carried out after vagotomy because of obstruction.

Methods of Study

Questionnaire

In the fall of 1952, questionnaires were sent to the 132 patients who had had vagotomy. To date, all but 6 have been heard from (Table 1). Of these 126, almost half had a personal interview, an x-ray examination or a gastric test of secretory function. Some had all three. A questionnaire unsupported by an interview or x-ray examination, may be misleading. The normal optimism of the average man prompts him to belittle his complaints and encourage his surgeon. But close examination of these questionnaires revealed tell-tale discrepancies.* In most cases of recurrence, the patient had professed to symptoms that raised our suspicions. The questions were designed with several purposes in mind:

Table 1. *Details of Ten-Year Follow-up Study after Vagus Resection.*

Type of Operation	No. of Patients	Patients Replying to Questionnaire	Patients Seen in Personal Interview	Patients Given Gastric Tests or X-rays
Vagotomy alone	82	77	33	32
Vagotomy combined with posterior gastroenterostomy	36	35	19	18
Previous gastroenterostomy	20	20		
Simultaneous operation	12	12		
"Late" gastroenterostomy	4	3		
Vagotomy combined with subtotal gastrectomy	14	14	2	2
Totals	132	126	54	52

To disclose patients in whom a suspicion of ulcer existed (they could then be brought to the laboratory for interviews and tests and gastrointestinal x-ray films).

To ascertain, in the large group without ulcer symptoms, how patients were getting along, with special reference to their state of nutrition, diet and abdominal complaints.

To determine what effect vagotomy had had on the type and amount of work each patient could do.

To discover whether these patients were taking medicines or if they had been under a doctor's care for gastrointestinal symptoms.

To find out what the patient thought of his operation.

The vagotomy group as a whole has turned out to be a cohesive one. Frequent return appointments to the gastrointestinal clinics and to doctors' offices, along with a large number of tests from time to time in the postoperative period, prompted them to be "doctor conscious." A large-scale re-evalua-

*An example is the patient who reports that he is fine in every way and well satisfied but who is subsequently found to be taking alkali, Banthine, bentyl and milk and cream frequently for epigastric discomfort.

tion of this same group in 1948[10] emphasized the need for follow-up study and now gives a measure of what time has done to the initial results.

Interview

Fifty-four patients were seen in a personal interview during which searching questions were put to them. These were carried out in some cases in conjunction with x-ray examination or studies of gastric secretion.

X-ray Studies

Forty patients have had gastrointestinal series within the past six to twelve months. These examinations were carried out to search for recurrent ulcer, and to evaluate the changes in gastric motility produced by time.

Gastric Secretory Tests

Twenty-four patients had gastric analyses combined with insulin gastric tests. These studies included free and total acid and determinations of chloride concentration. Twenty-seven patients had studies of uropepsin output.[19]

For several reasons the results of these few tests do not reflect the true status of the group. The number of tests is too small for statistical purposes. It has been shown repeatedly that recurrences of ulcer after surgery do not correlate well with any findings in the laboratory.[20] The finding of a flat insulin gastric test and a negative test for free acid on fasting does not ensure against recurrence. Likewise, a high free acid and rise in acid after insulin is not necessarily indicative of the presence or likelihood of development of an ulcer.

Results

Questionnaires, Interviews and X-ray Examinations

Pure vagotomy (transthoracic). We consider that a patient has an excellent result if he has had no ulcer pain or bleeding, no vomiting and no sensation of severe fullness after meals; if he can eat three regular meals a day; if he has lost no time from work; and if he honestly regards the result as close to perfect.

The result is regarded as satisfactory if the patient has only occasional episodes of abdominal distress (not "ulcer pain"); if he has considerable, but not overwhelming, difficulty with fullness after eating; if he has to watch what he eats; if he has one or two episodes of vomiting a year; if he takes occasional medicines; if he has minor bowel problems; if he has to stay out of work one or two days a year; and if he honestly considers himself free of the ulcer problem but regards the result as not perfect.

There were also a few patients with definite symptoms but without proof of recurrent ulcer, or with symptoms due to the side effects of vagotomy that were troublesome. These two groups are classified as borderline between success and failure, and the results are considered dubious.

The rest are regarded as complete failures because of proved recurrent ulcer, bleeding indicative of ulcer or incapacitating side effects of vagotomy.

Table 2. *Results, Short of Failure, of Vagus Resection after Ten-Year Follow-up Study.*

Operation	Total Patients	Patients with Excellent Results	Patients with Satisfactory Results	Patients with Vagal Symptoms	Patients with Ulcer Symptoms	Patients Who Later Died*
Vagotomy alone	82	31	21	3	6	3
Vagotomy combined with posterior gastroenterostomy	36	17	9	1	1	2
Vagotomy combined with subtotal gastrectomy	14	10	2	1	0	1
Totals	132	58	32	5	7	6

*Not as a result of either ulcer or operation.

In this group of 77 cases (Table 2)* the results were classified as excellent in 31, or 40 per cent.† The results in 21, or 27 per cent, were classified as satisfactory.

There were 9 patients in the dubious group. Three had postvagotomy symptoms of a severe degree; 6 had ulcer symptoms but no proof to date of recurrence. Three patients in the pure vagotomy group have died, none of ulcer complications and none of surgical complications.

In 19 patients (25 per cent) the results were classified as failures (Table 3). Three had bleeding, although no ulcer could be shown by gastrointestinal series; 10, or 13 per cent,‡ had recurrent ulcers. Two had uncontrollable symptoms of obstruction and had a subtotal gastrectomy; in 4 with obstructive symptoms a gastroenterostomy was performed, and these 4 were then incorporated in the group of vagotomy combined with posterior

*A certain number of these patients were interviewed. The results in others were classified as excellent after careful evaluation of their status in the 1948 follow-up study, as well as at the present, so that more often than not, a long-term, frequently repeated evaluation was possible over a period of six to ten years.

†Standard deviation 3.8.

gastroenterostomy. In all, subtotal gastrectomy was carried out after pure vagotomy in 11 cases.

Five patients have not been heard from.

In terms of vagus side effects, these patients have done only moderately well. The vast majority complain of fullness after eating, which reflects the status of gastric motility after vagotomy. For some this compromises daily activities to a considerable degree. In a very few, these symptoms have been almost incapacitating, leading to constant eructation, regurgitation and occasionally vomiting. In a few others, as noted above, these symptoms, particularly when associated with a stenosing duodenal-ulcer scar, have necessitated gastroenterostomy or subtotal gastrectomy. The frequency of postvagotomy diarrhea is now almost nil. One or two patients state that they still have short intermittent episodes of looseness of the bowels, but nothing of any severity. Many have had this complaint, now abated, for two to five years.

The majority of this group have either remained stationary in weight or gained a few pounds. Most patients have continued with their smoking and drinking habits despite strong criticism from the doctor.

Economically speaking, none of the patients in the excellent or satisfactory group have lost time from work. Nor have any been forced to take on easier jobs or shorter hours. Those with results considered failures and some of those with borderline results between failure and satisfactory have lost varying amounts of time from work.

A few patients are still taking medicines. Most of these are in the group of whom we are suspicious of further trouble. Those in the excellent and satisfactory groups on the whole do not resort to medicines for digestive reasons.

It is apparent that the 40 per cent of results classified as excellent and the 27 per cent classified as satisfactory are good from what certainly can be described as the simplest, least traumatizing surgical procedure available today for duodenal ulcer. This in itself is a particularly interesting finding. Our knowledge of the timing of recurrence suggests that most of these 52 patients will continue to do well since they have remained well for six to ten years.

Vagotomy with posterior gastroenterostomy. The present-day acceptance throughout this country of the "failure" of vagotomy alone for the treatment of duodenal ulcer places special importance on the analysis of the results of vagotomy combined with posterior gastroenterostomy. The number of cases is small when compared with those in which vagotomy alone was performed. The group is further divided into 20 patients with previous gastroenterostomy (Table 1), who were admitted to the Massachusetts General Hospital between 1943 and 1948 with marginal ulcers and in whom vagotomy was then performed. There were 12 patients who had vagotomy and gastroenterostomy carried out simultaneously. The grouping is further confused by the addition of 4 patients who had "late" posterior gastroenterostomy to overcome postvagotomy pyloric obstruction. We appreciate the fact that this group lacks uniformity for these reasons and also that patients in whom a marginal ulcer has once developed may not respond to vagotomy in the same manner as those who have had an initial vagotomy with gastroenterostomy. Nevertheless, the fundamental anatomic arrangement is the same in all cases, and our later interpretations are made with this consideration in mind.

TABLE 3. *Data in Patients with Results Regarded as Failures.*

OPERATION	PATIENTS WITH BLEEDING WITHOUT ULCER	PATIENTS WITH VAGAL SYMPTOMS	PATIENTS WITH RECURRENT ULCER	PATIENTS WITH SUBTOTAL GASTRECTOMY	PATIENTS WITH POSTERIOR GASTROENTEROSTOMY (LATE)
Vagotomy alone	3	2	10 (13%)	11	4
Vagotomy combined with posterior gastroenterostomy	2	—	5 (14%)	2	—
Vagotomy combined with subtotal gastrectomy	0	—	1 (7%)	—	—
Totals	5	2	16	13	4

The results were classified as excellent in 17 patients (50 per cent) in this group. In 9 additional patients (25 per cent) results were regarded as satisfactory. Two patients had moderately severe vagal symptoms. One now has ulcer symptoms without x-ray evidence of recurrence. There were no operative deaths. Two patients died of causes unrelated to duodenal ulcer. One patient did not return his questionnaire.

There were 7 failures in the group (20 per cent). Two patients had serious bleeding, with no x-ray proof of ulcer as of this writing. Five patients (14 per cent*) had recurrences proved by operation, pathology specimen or x-ray examination and, in 1 case, by gastroscopy. In 2 cases the recurrence required subtotal gastrectomy. One of the 3 remaining recurrences was controlled fairly well medically, the patient dying from an unrelated cause in 1951. One patient has a large gastric ulcer recurrence at present, and another had a jejunal

*Standard deviation 5.9.

ulcer in 1950 that subsequently healed with medical management.

Vagotomy and subtotal gastrectomy. The present series of cases is small, comprising 14 in which vagotomy was carried out because of marginal ulcer after subtotal gastrectomy. For this reason, perhaps they represent a particularly virulent group in which there is less hope of permanent cure. The results in 13 of these patients can now be classified as excellent. One patient died of unrelated causes, and was found to have a gastric ulcer on the lesser curvature at autopsy.

TABLE 4. *Laboratory Findings in Patients with Recurrent Ulcer.*

PATIENT	PREVIOUS ULCER	OPERATION	YEAR	FASTING FREE HYDROCHLORIC ACID*	INSULIN GASTRIC TEST†	UROPEPSIN	TYPE OF RECURRENT ULCER	YEAR	REMARKS
Ros.	Duodenal	Vagotomy	1946	Low — rise with histamine in 1947; low in 1952.	Slight rise in 1947; flat after 4 hr. in 1952.	—	Duodenal	1951	Recurrent ulcer developed in 1951; subtotal gastrectomy in 1952 for pyloric obstruction.
Mor.	Duodenal	Vagotomy	1947	High normal — rise with histamine in 1949	Flat after 4 hr. in 1949	High	Duodenal	1949	Subtotal gastrectomy in 1949 for active duodenal ulcer
Med.	Marginal	Vagotomy; posterior gastroenterostomy (old).	1946	Low in 1950	Sharp rise in 1947; flat after 4 hr. in 1950	—	Marginal	1950	Subtotal gastrectomy in 1950 for active marginal ulcer
Osb.	Marginal	Vagotomy & posterior gastroenterostomy	1947	Low in 1948	Flat after 4 hr. in 1948	—	Marginal	1948	Marginal ulcer seen at gastroscopy in 1948; patient now well.
Fay.	Marginal	Vagotomy; posterior gastroenterostomy (old).	1947	Low in 1953	Slight rise in 1953	High	Gastric	1953	Patient now under treatment
Pas.	Duodenal	Vagotomy	1947	High in 1953	Sharp rise in 1953	High	Duodenal	1953	Patient now under treatment
Rud.	Duodenal	Vagotomy	1948	High normal in 1950	Not done	—	Duodenal	1950	Subtotal gastrectomy for active duodenal ulcer in 1951
Mora.	Duodenal	Vagotomy	1946	Normal in 1947	1 test flat in 1947; 1 test late rise in 1947.	—	Duodenal	1947	Subtotal gastrectomy for active duodenal ulcer in 1947
Sil.	Gastric	Vagotomy	1946	Low	Flat in 1947; flat in 1948.	—	Gastric	1948	Subtotal gastrectomy for active gastric ulcer
Cha.	Duodenal	Vagotomy	1946	None — rise with histamine in 1952	Not done	—	Pyloric	1952	Subtotal gastrectomy in 1952
Smi.	Duodenal	Vagotomy & posterior gastroenterostomy	1947	Low in 1948	Rise in 1948	—	Marginal	1949	Ulcer pain and hematemesis in 1949; patient died of stroke in 1952.
McN.	Duodenal	Vagotomy	1946	Low in 1949	Rise in 1949	—	Duodenal	1949	Subtotal gastrectomy for ulcer in 1949
Cac.	Marginal	Vagotomy; subtotal gastrectomy (old).	1952	Low in 1952	Flat in 1952	High	Marginal	1952	Recurrent marginal ulcer developed 4 mo. after vagotomy; now healed with medicines.
Eng.	Duodenal	Vagotomy	1944	Normal in 1949	Sharp rise in 1949	—	Duodenal	1949	Subtotal gastrectomy in 1951

*Normal free acid considered to be between 15 and 25 clinical units.

†A positive insulin test is considered to be one in which the fasting free acid rises 20 units or more on stimulation. A positive response in a patient with no fasting free acid is a rise of at least 10 units after stimulation.

X-ray Examination

Gastrointestinal examinations were carried out in most of the cases listed as failures. An additional 40 were performed on patients with results regarded as excellent and satisfactory. When recurrent ulcers have been seen, no clear-cut correlation with gastric motility has been possible. Patients with vagotomy alone revealed old chronic ulcers with scarring, varying amounts of duodenal deformity or pseudodiverticulum formation, and varying amounts of stasis. Those who had had vagotomy with gastroenterostomy now show scarring also, varying amounts of emptying through the pylorus and normally functioning stomas. It can be said, however, that the striking finding in the postvagotomy stomach is a beginning return of motility and tone. Whether this represents a "regeneration" of vagal fibers or an assumption of vagal function by other pathways is an open question.

Tests of Gastric Secretion

In the spring of 1953 we set up a program of study that to date has been carried out on 24 patients with the following aims in sight:

To analyze the basal, fasting free acid, total acid and chloride concentration levels in patients with pure vagotomy. (A few cases of vagotomy combined with posterior gastroenterostomy were included in the study.)

To ascertain the response of the parietal cell and of the gastric chloride concentration to insulin hypoglycemia.

To evaluate the basal uropepsin levels in patients after vagotomy and to evaluate the response of uropepsin to gastric stimulation.*

The tests on the patients who had already had recurrences failed to reflect any consistent pattern so far as basal and stimulated levels of free and total acid are concerned (Table 4). Some showed higher than normal and others quite normal basal acid levels. In some cases a rise of moderate proportions followed insulin hypoglycemia, but none showed the dramatic peak of acid secretion characteristic of the ulcer patient before operation. Others had perfectly flat insulin gastric curves.†

Table 5 shows the results of the tests in the group studied in 1953. The effect of vagotomy was considered successful in most of these patients.

The studies consisted in passage of a Levin tube into the fasting stomach after which determinations of baseline free and total acid and chloride concentration were obtained. Insulin (15 units) was then administered intravenously. Blood samples for sugar determinations were drawn, and gastric samples obtained at half-hour intervals, the tube being clamped between aspirations. Multiposition aspiration was used at the close of each half-hour period to drain the stomach as completely as possible. The aspirations were continued for a total of two and a half hours. We appreciate the inadequacy of simple aspiration in determining the total rate of hydrochloric acid output. We have used the single aspiration only as a guide to free and total acid levels.

Fasting levels of free acid were normal or low (less than 20 clinical units) in the majority of patients with pure vagotomy studied. There were 7 patients in this group who had high levels of free acid (over 20 clinical units) on fasting. One of these was a patient with a recurrent active duodenal ulcer crater. Three were patients who complained of abdominal distress not unlike their old ulcer pain, but whose gastrointestinal x-ray examinations were negative for recurrent ulcer. The remainder had higher than normal levels of fasting free acid, with no clinical or x-ray evidence of ulcer recurrence or other gastric difficulty.

The fasting acid levels in any patient with a gastroenterostomy are notoriously low so that their accurate interpretation is difficult. The patient in this group with a recurrent gastric ulcer had no free acid on fasting.

The response of the normal, intact stomach to insulin hypoglycemia consists of a rise in free and total acid levels beginning about three quarters of an hour after the injection. This rise follows the lowest blood sugar levels by approximately half

TABLE 5. *Laboratory Findings in Patients without Evidence of Recurrent Ulcer.*

PATIENT	PREVIOUS ULCER	OPERATION	YEAR	FASTING FREE HYDROCHLORIC ACID	INSULIN GASTRIC TEST	URO-PEPSIN	REMARKS
Lev.	Duodenal	Vagotomy	1946	Normal in 1951	Late rise after 4 hr. in 1951	—	Gastrointestinal series negative for ulcer in 1951 & 1953; ulcer-like pain in 1951; no pain in 1953.
Hei.	Duodenal	Vagotomy	1945	Normal in 1948; high in 1953.	Flat in 1948; flat after 3 hr. in 1953.	High	Symptoms of pyloric obstruction; no ulcer on gastrointestinal series.
Ros.	Duodenal	Vagotomy	1947	Low in 1948	Flat in 1948	High	Patient has heartburn and fullness after eating; x-ray examination negative.
Gal.	Duodenal	Vagotomy	1947	Low in 1947; high in 1953.	Flat in 1947; flat in 1953.	High	No complaints; x-ray examination negative.
Sla.	Marginal	Vagotomy	1945	Low in 1947; low in 1953.	Flat in 1947; late rise in 1953.	High	Patient has had bleeding indicative of recurrent ulcer but no x-ray evidence
Sm., W.	Duodenal	Vagotomy	1946	Normal in 1953	Flat in 1953	High	Patient well; x-ray film normal.
Sca/	Marginal	Vagotomy	1948	Low in 1953	Rise in 1953	Normal	Patient feels very well; x-ray film normal.
Mah.	Duodenal	Vagotomy	1947	Normal in 1953	Flat in 1953	High	Patient well; x-ray examination negative.
Iom.	Duodenal	Vagotomy	1946	Low in 1953	Flat in 1953	—	Patient well; x-ray examination negative.
Jol.	Duodenal	Vagotomy	1946	High normal in 1953	Flat in 1953	High	Patient has recurrent ulcer-like pain, but gastrointestinal series shows no ulcer
Don.	Duodenal	Vagotomy	1945	Low in 1953	Rise in 1953	Normal	Some bleeding; gastrointestinal series negative for ulcer recurrence.
Wal.	Duodenal	Vagotomy	1946	Low in 1953	Rise in 1953	—	Patient well; x-ray examination negative.
Pai.	Duodenal	Vagotomy	1946	Low in 1948; low in 1953.	Flat in 1948; flat in 1953.	High	Patient well; x-ray examination negative.
Mat.	Duodenal	Vagotomy	1947	Low in 1953	Flat in 1953	—	Patient well; x-ray examination negative.
Mar.	Duodenal	Vagotomy	1946	Low in 1951	Flat in 1951	Normal	Patient well; x-ray examination negative.
Cot.	Duodenal	Vagotomy	1946	Low in 1953	Rise in 1953	—	Occasional "ulcer"-like pain; gastrointestinal series negative for ulcer.
Pic.	Duodenal	Vagotomy	1948	Low in 1953	Rise in 1953	—	Patient well; x-ray examination negative.
Sto.	Duodenal	Vagotomy	1947	High in 1953	Rise in 1953	—	Some pain in "ulcer" area; x-ray examination negative.
Hay, A.	Duodenal	Vagotomy	1945	Low in 1948; high in 1952.	Flat in 1948; flat in 1952.	Normal	Patient fairly well; no ulcer symptoms; x-ray examination negative.
McI.	Duodenal	Vagotomy and posterior gastroenterostomy	1947	High in 1950	Flat in 1950	—	Patient well; x-ray examination negative.

*Gray and others showed that the uropepsin in the urine is in fact a direct reflection of the amount of pepsinogen being released in the stomach. In more accurate terms, it has been shown that pepsinogen is absorbed into the blood stream in small amounts before hydrochloric acid has had a chance to activate it. The amount absorbed approximates 1/100th of the pepsin actually produced in the stomach, and the amount in the urine is an exact reflection of change in pepsin production in the stomach. As might be expected, uropepsin levels are high in patients with ulcer disease, and low in patients with gastric carcinoma or pernicious anemia.[13]

†We have seen a variety of combinations. One patient had a recurrent ulcer crater despite a perfectly flat insulin gastric test. Another had a recurrent ulcer with evident continuing hypomotility on x-ray examination. It is of great importance that ulcer recurrence may be observed in the face of a maintenance of the measurable physiologic effect of vagotomy. This leads, of course, to further speculation concerning other etiologic pathways.[8]

an hour. The highest level of acid occurs one to one and a half hours after insulin injection and then slowly returns to normal. The occurrence of "a late response" to hypoglycemia (two to four hours) seen in some of the vagotomy patients has stimulated further thought whether or not pathways other than vagal (? hormonal) are involved.[22]

There was no acid response to insulin hypoglycemia in the majority of cases six to ten years after vagotomy alone. However, 6 patients showed a classic early rise in free and total acid, and 2 a late rise. Of these 8 patients, 1 now has a recurrent duodenal ulcer crater, and 2 have occasional episodes of epigastric "distress" that raise suspicion of activity. The others are symptom free, without evidence of ulcer difficulty. None of these tests lasted longer than two and a half hours so that other late acid rises may have been missed.

The results after insulin in the cases in which vagotomy was combined with posterior gastroenterostomy were varied but impossible to interpret owing to the small number of patients studied. The patient who now has a recurrent gastric ulcer showed a late acid rise after administration of insulin.

TABLE 6. *Gastric Chloride Concentrations before and after Insulin Administration.**

SUBJECT	GASTRIC CHLORIDE CONCENTRATION				
	FASTING	30 MIN. AFTER INSULIN	60 MIN. AFTER INSULIN	90 MIN. AFTER INSULIN	120 MIN. AFTER INSULIN
	milliequiv. / liter	*milliequiv. / liter*	*milliequiv. / liter*	*milliequiv. / liter*	*milliequiv. / liter*
Ma.	83.6	55.0	137.0	137.0	137.2
Sc.	101.2	88.2	124.5	129.8	118.8
Jo.	119.1	105.2	116.9	102.4	112.0
Fa.	108.0	94.6	132.8	138.0	126.7
St.	115.5	108.9	131.6	138.0	126.5
Wa.	126.8	92.4	149.2	154.0	136.5
Pico.	129.1	65.0	123.2	121.0	—
Do.	96.2	92.4	108.8	134.2	140.7
Co.	116.2	85.4	125.7	102.1	92.6
Mar.	103.3	88.6	—	—	100.3
Pa.	117.7	130.4	146.8	134.9	146.8
Jo.	100.1	131.4	157.3	126.2	115.5
Sm.	103.1	106.7	100.1	82.8	91.5
Mat.	113.2	116.3	122.8	123.9	104.0
Pi.	100.2	112.4	127.2	—	140.1

*15 units injected intravenously.

The chloride concentration level, although not as important an index of acid secretion as the chloride ion output per unit of time,[11] reflects the level of parietal-cell activity. Chloride concentrations tend to be low in people with achlorhydria and high in patients with active peptic ulcer (the normal range is 85 to 120 milliequiv. per liter).

In the small group of 15 postvagotomy patients studied to date, the fasting chloride concentration levels averaged 108.9 milliequiv. per liter (Table 6). Ten patients showed a drop in chloride concentration half an hour after insulin injection to an average level of 87.6 milliequiv. per liter. The other 5 demonstrated no significant change or a slight rise. For the entire group the average rise, an hour after administration of insulin, was 19.7 milliequiv. per liter.

These data suggest that chloride concentration levels in the postvagotomy stomach are normal but that changes in chloride concentration may occur with stimulation.

The uropepsin data* from urine samples collected over a twelve-hour period are presented in Table 7. The majority fall within the ulcer range (over 1500 units per twelve hours). The average uropepsin output after vagotomy is 2234 units per twelve hours.[19] This is lower than the ulcer mean (3000 units per twelve hours), but higher than normal (<1500 units per twelve hours) and than the mean for patients with subtotal gastrectomy (1400 units per twelve hours).

TABLE 7. *Uropepsin Data.*

SUBJECT	RANGE *units/12 hr.*
Normal person	750–1500
Patient with ulcer	1500–3500
Patient after vagotomy (mean)	2234
Patient after gastrectomy (mean)	1400

DISCUSSION

There appears to be no clear-cut correlation between laboratory findings of gastric secretion and clinical findings of well-being. There may be a complete lack of correlation between acid levels, insulin response and ulcer recurrence. There also appears to be no correlation between low levels of gastric acidity and flat insulin curves, nor between high gastric acidity and rising insulin curves.

However, there does appear to be a trend of change in gastric secretion and function as time passes. Acid levels are higher in more of the patients after vagotomy than they were five years ago; there are more positive insulin tests[1]; there is x-ray evidence of returning gastric motility in more of the patients. Uropepsin levels fail to find their normal range after vagotomy. It appears that pepsin may be more prominent than hydrochloric acid in the production of ulcer. This finding and the trend of secretory levels noted above shed new light on the role of the vagus. Does the vagus control hydrochloric acid secretion to a greater degree than it does pepsin? Does vagotomy in fact effect any radical change in pepsin production? Do the recurrent ulcers represent the end result of "partial escape" from vagotomy and its effect on acid production in addition to an already present substantial level of pepsin? Continued investiga-

*We are indebted to Dr. Seymour J. Gray for the uropepsin determinations done in his laboratory.

tion in the laboratory with patients who have had vagotomy may help to answer these questions.

Although the physiology of gastric secretion and its study are important the final criterion of success or failure in the treatment of an ulcer lies in the clinical evaluation of the patient some time after his definitive surgical procedure.

In 1949 we completed a study of end results in duodenal-ulcer therapy.[12] One of the purposes of that study was to provide a broad background against which other forms of therapy could be viewed in perspective. Reference to these data on subtotal gastrectomy (175 cases) reveals a number of interesting points in comparison with the vagotomy series (vagotomy alone and vagotomy with gastroenterostomy). The mortality, as noted above, was consistently higher after subtotal gastrectomy. In terms of morbidity, recurrent ulcer and the need for subsequent surgery, the data at that time showed that vagotomy failed to hold its own with subtotal gastrectomy.

On the basis of the data presented here it is even clearer that vagotomy alone no longer has a place in the treatment of uncomplicated intractable duodenal ulcer. It has failed primarily because it has not prevented recurrent ulceration and because subtotal gastrectomy has a significantly higher promise of success.

Nevertheless, there is something appealing in the simplicity of section of the vagus nerves, which is capable of curing very intractable ulcer disease in 67 per cent of the patients. Here, then, is an operation with a minimal mortality that can relieve ulcer pain and heal an ulcer crater. The basic defect doubtless lies in our inability to select accurately the patients whose disease is adapted to this approach.

The results in the small series of patients who have had vagotomy combined with gastroenterostomy are disappointing. There have been 5 recurrent ulcers divided between patients who had gastroenterostomy as a separate procedure before vagotomy and those who had gastroenterostomy at the time of or after vagotomy. Patients coming to vagotomy with a pre-existing marginal ulcer may well be classified as a group with virulent ulcer. Whether or not the addition of a much larger number of cases of vagotomy and gastroenterostomy done simultaneously would alter the recurrence rate in this group is hard to say. It seems likely, however, that as time passes, the expected tendency for a marginal ulcer to develop in a patient with a gastroenterostomy will increase, and this will not be counteracted adequately by a concomitant section of the vagus nerves. Gastric digestive function seems to escape the effect of vagus resection as time passes, possibly because other gastric secretory mechanisms go into action. The digestive function is a primitive property and not easily discouraged. Its abnormal activity produces ulcer. Between 1900 and 1935, surgeons learned that when jejunal juice and gastric juice were brought into contact surgically, the stomach too often won out, ulcerating the jejunum rather than yielding to its alkalinizing effect. We find no evidence here that vagotomy will alter that basic relation, though it may postpone the evil day of reckoning.

The results in the small group of patients who have had subtotal gastrectomy followed by vagotomy are of interest. The concept of combining gastrectomy with vagotomy has several advocates.[13, 15] Determination whether or not the magnitude of surgery involved is warranted on the basis of ulcer results must await the passage of time.

Vagotomy has a definite place in the treatment of marginal ulcer occurring after subtotal gastrectomy.

Out of the confusion of the laboratory studies done to date, the following secretory findings appear to be substantiated:

It is possible for a patient to remain free of symptoms and of x-ray evidence of ulcer recurrence for five to ten years despite a positive insulin gastric test and high free acid levels (Table 5).

It is possible for a marginal ulcer to develop in the face of a flat four-hour insulin test and low free acid values (Table 4).

There are patients who still have flat insulin tests eight or nine years after vagotomy.

With the passage of time, the incidence of positive insulin gastric tests is increasing.

With the passage of time, there is a comparable increased incidence of higher free acid values in the fasting samples.

Vagotomy seems to produce only a minor change in uropepsin levels, which remain within the ulcer range eight to ten years after vagotomy.

Summary and Conclusions

Vagotomy alone is not a satisfactory primary surgical procedure for duodenal ulcer when competing with subtotal gastrectomy carried out in competent hands and with a low mortality.

The efficacy of vagotomy combined with posterior gastroenterostomy in the treatment of duodenal ulcer is not supported by our data. Judgment must be reserved because of the high incidence of pre-existent marginal ulcer in our small series. More important, however, is the fact that *recurrent ulcer may occur while physiologic effects of vagotomy persist.* This bodes ill indeed for the patient who has had primary vagotomy combined with gastroenterostomy for duodenal ulcer.

The place of vagotomy in the treatment of marginal ulcer after subtotal gastrectomy is substantiated.

The results of present-day laboratory tests have no consistent correlation with clinical results in the evaluation of surgery for duodenal ulcer.

The patients studied were operated on for the most part at the Massachusetts General Hospital by Dr. Moore and by other surgeons of the staff. We are indebted to the Surgical Service and the Hospital for extensive collaboration in this follow-up study.

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SURGICAL CARE OF COMPLICATED GASTRIC AND DUODENAL ULCER IN SMALL HOSPITALS*

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THIS report is concerned with the surgical care of gastric and duodenal ulcers in two small hospitals in New Hampshire. It covers a period of two and a half years, ending June 30, 1953, during which gastric resection for gastric, duodenal or gastrojejunal ulcer was performed in 103 cases.

Table 1. *Indications for Gastric Resection for Ulcer.*

Indication	Cases of Gastric Ulcer	Cases of Duodenal Ulcer	Cases of Marginal Ulcer	Totals	Percentage
Massive hemorrhage	1	11		12	11.7
Bleeding	7	23		30	29.1
Obstruction		10		10	9.7
Intractability	12	35	4	51	49.5
Totals	20 (19.4%)	79 (76.6%)	4 (3.9%)	103	

The types of ulcer and primary indications for resection are presented in Table 1. Intractability includes failure of either a gastric or duodenal ulcer to heal on medical therapy, persistent pain or bleeding on a good medical program and repeated perforation.

The circumstances of gastric resection are outlined in Table 2. The word "emergency" is well established in surgical parlance and hospital usage. It is not a good term, psychologically or in lay tradition, and a better one should be found. It too often indicates that an unanticipated, unprepared-for situation is met under conditions of physical stress and emotional tension by whoever is on the scene, and with whatever mediums or equipment happens to be at hand. Emergencies in this meaning probably should never occur in a well run modern hospital, and they are certainly not a tolerable feature in the proper management of massive gastrointestinal hemorrhage. This is not an uncommon disease. The small hospital, as well as the

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large one, should have a plan of action for treating upper gastrointestinal hemorrhage. If rapid therapy is needed, it can then still be methodical and efficient. The immediate operative procedure itself, when required, should be emergency only in the sense that it cannot be scheduled at a chosen time in the future, but the same facilities, co-ordinated teamwork and efficiency should apply as though the operation had been scheduled electively.

The average and median ages for different categories of cases are shown in Table 3. The patients were predominantly of middle age, but 9 (8.7 per cent) were over seventy, and 8 (7.8 per cent) were under thirty years of age. The average age of patients with resection for gastric ulcer was twelve years higher than that of those with duodenal ulcer.

Table 3. *Age Distribution in Various Classifications.**

Classification	No. of Cases	Mean Age	Median Age
		yr.	yr.
Elective operation	91	49.6	51.0
Emergency operation	12	51.6	52.0
Duodenal ulcer	79	46.7	46.0
Gastric ulcer	20	58.8	57.5
Marginal ulcer	4	65.8	67.5

*The youngest patient was 25, and the oldest 94 years of age; 8 patients (7.8 per cent) were under 30, and 9 (8.7 per cent) were over 70 years of age.

Massive Hemorrhage

Twelve cases, or 11.7 per cent of all operations for ulcer, were emergency procedures. There is an impressive parallelism between duration of symptoms and occurrence of massive hemorrhage (Table 4). Eight, or two thirds of the 12 patients requiring emergency operation, had symptoms of more than three years' duration. Beyond the eighth year, the percentage of emergency cases requiring resection was consistently higher than that of the elective group. However, 1 patient with no previous history had a massive hemorrhage requiring emergency resection; this patient was forty-nine years of age.

Age of Patients

Seven patients (58.3 per cent) with massive hemorrhage were over fifty, and 3 (25 per cent) over seventy years of age (Table 5). The average age of 4 patients requiring emergency operation who had symptoms of less than three years' duration was forty years, 1 being over fifty, and that of 8 patients requiring emergency operation who had symptoms of over three years' duration was fifty-five, 2 being under thirty. Although there were no deaths in this group of 12 emergency resections, one can assume that some chronically ill elderly patients are going to die from massive hemorrhage, even though they have emergency resection to control bleeding and receive the best of modern surgical care. In this small group of cases, it is obvious that patients with symptoms of longest duration who are over fifty years of age (and consequently more often subject to cardiovascular and renal problems) are most likely to present the additional complication of massive, life-endangering hemorrhage.

Five of 12 elective resections in cases with symptoms of less than two years' duration were also done for bleeding as the primary indication. Of the 91 patients with resections under elective circumstances, the indication was bleeding in 30. Preparation in these cases was mainly a matter of restoring blood volume before operation.

In 10 cases the indication for elective surgery was obstruction. There was no emergency resection for obstruction. As is well known, complete obstruction of the gastric outlet that fails to respond temporarily and at least partially to suction and alkalis and rest is probably caused by carcinoma. Acute obstruction from cicatricial contracture, or from edema about an active ulcer, rarely remains complete under therapy, although it may remain functionally intol-

Table 2. *Circumstances of Resection.*

Type of Procedure	Cases of Gastric Ulcer	Cases of Duodenal Ulcer	Cases of Marginal Ulcer	Totals	Percentage
Emergency	1	11		12	11.7
Elective	19	68	4	91	88.3
	20	79	4	103	

Table 4. *Massive Hemorrhage and Duration of Symptoms.*

Type of Procedure	No. of Cases	Incidence of Massive Hemorrhage					
		Duration of 3 yr.	Duration of 8 yr.	Duration of 10 yr.	Duration of 15 yr.	Duration of 20 yr.	Duration of 30 yr.
		%	%	%	%	%	%
Emergency	12	66	50	42	25	17	8
Elective	91	26	46	38	17	13	5

erable, and it is in such cases that obstruction is listed as the indication for operation.

Massive hemorrhage from a peptic ulcer requiring emergency gastric resection has been defined in various ways. We have tried to follow the generally accepted definition. As applied in our practice for several years and in this report, massive upper gastrointestinal hemorrhage means blood loss causing the hemoglobin level to fall below 9 gm. per 100 cc., accompanied by alteration in vital signs indicative of shock, and failure to elevate the hemoglobin level and stabilize the vital signs by administration of 1500 to 2000 cc. of blood within a twenty-four-hour period. The average level of hemoglobin on admission in the emergency cases was 7.6 gm., the highest 9.6 gm., and the lowest 4.6 gm. per 100 cc. (Table 6). The average amount of blood given in these twelve cases from admission to operative control of the bleeding was 3500 cc., the largest amount being 8000 cc. and the smallest 1500 cc.

Hemoglobin level on admission and associated signs of shock are not in themselves indications for emergency resection. In the cases in which elective operations were done, the average hemoglobin reading was 10.5 gm. per 100 cc.; 3 patients had levels below 6 gm., and 12 levels of 9 gm. per 100 cc. or less. The difference is that the bleeding stopped or slowed down so that the hemoglobin level could be raised, the vital signs stabilized, and operation done electively.

Table 5. *Age of Patients in Relation to Duration of Symptoms.*

Type of Procedure	No. of Patients	Average Age of Patients with Symptoms for Less than 3 Yr.	Average Age of Patients with Symptoms for More than 3 Yr.	Age over 50 Yr.	Age over 70 Yr.
		yr.	yr.	no. of patients	no. of patients
Emergency	12	40	55	7 (58%)	3 (25%)
Elective	91	58	49	49 (54%)	6 (7%)

Table 6. *Hemoglobin Levels and Amount of Blood Given.*

Type of Operation	Hemoglobin Level on Admission			Amount of Blood Given		
	average gm./100 cc.	highest gm./100 cc.	lowest gm./100 cc.	average cc.	highest cc.	lowest cc.
Emergency	7.6	9.6	4.6	3500	8000	1500
Elective	10.5	—	5.0	—	—	—

Technical Considerations

Judged solely by ability of the pathologist to find the ulcer in the resected specimen, the ulcer was removed in 78 cases (Table 7), including all cases of gastric ulcer. Of the remaining 25, the ulcer was excised from the duodenum in all except 4, which lay in the second portion, on or about the common duct. No effort was made to remove these ulcers; we have tried to leave lesions involving the pancreas from the proximal duodenum in situ, after mobilizing the duodenum, to lessen the risk of pancreatitis. Also, in spite of the fact that a specimen is thereby lost for pathological examination, we have not hesitated to put clamps across and transect through an ulcer in the first portion of the duodenum after mobilization because of the longer duodenal stump thus safely obtained for inversion. So far as we know, duodenal ulcers have no malignant potential.

Seventy-five per cent of the stomach was resected in all cases. Vagotomy was added to the resection for the 4 gastrojejunal ulcers. Gastroenterostomy was of the short-loop retrocolic type in two thirds and of the long-loop antecolic type in one third of the cases.

Table 7. *Excision of Ulcer in Relation to Pathological Diagnosis.*

Pathological Diagnosis	Removal of Gastric Ulcer	Removal of Duodenal Ulcer	Removal of Marginal Ulcer
	no. of cases	no. of cases	no. of cases
Ulcer in specimen	20	54	4
No ulcer in specimen	—	25	—

No emergency resection was done for perforated ulcer nor was any done for obstruction. Massive, otherwise uncontrollable hemorrhage was the only indication for emergency resection.

The 4 gastrojejunal ulcers, 1 of which was associated with a colic fistula, were resected in one stage. Two resections were done as staged procedures because of severe inflammatory reaction about the duodenum. In no case was a catheter put into the duodenum. We wished that a catheter had been chosen for 1 patient (who subsequently died in uremia), because when the invasive duodenal ulcer was broken into, the base was found to contain the common and pancreatic ducts.

Complications

A number of nonfatal complications occurred (Table 8). The 4 pulmonary infarcts were in the 5 patients who had phlebitis. Elastic support to the legs was used prophylactically in these as in other cases. Treatment after infarction consisted of heparin administration every eight hours, controlled in amount by reference to serially determined blood clotting time (Lee-White method).

Table 8. *Complications.*

Complication	No. of Cases
Auricular fibrillation	1
Thrombophlebitis	5
Pulmonary infarction	4
Coronary thrombosis	1
Prostatic obstruction	1
Prolapsed hemorrhoids	1
Minor wound infection	1
Wound dehiscence	3
Major stomal obstruction	1

One major stomal obstruction occurred, and eventually a new anastomosis was necessary.

Deaths

There were 2 deaths. The first occurred in a fifty-nine-year-old man with a thirty-year history of intractability and bleeding. There had been two previous hospitalizations for bleeding, and one for myocardial infarction. He had cerebral arteriosclerosis and hypertension, and one kidney had previously been removed. At elective operation, the duodenum was too indurated to permit mobilization. The antral mucosa was resected, and the gastric pouch closed. Postoperatively, atelectasis, deep phlebitis and pulmonary infarction developed. Leakage from the gastric stump occurred.

The other patient who died was a seventy-two-year-old man with a thirteen-year history of recurring massive hemorrhages who could not accurately recall the number of previous hospitalizations. Over forty transfusions had been given. Ten years earlier, a posterior gastroenterostomy had been done without benefit. In addition to obstruction, he had large active duodenal and gastrojejunal ulcers. At operation, both ulcers were resected. The duodenal ulcer had eroded about the common duct and pancreatic ducts into the head of the pancreas. A postoperative pancreatic fistula developed. The patient died in uremia on the nineteenth postoperative day.

Discussion

Surgical care in small hospitals should be the equivalent of that in larger institutions. Trained physicians and surgeons, adequate hospital equipment, supplies and facilities are needed. Many towns in New England meet these requirements; others do not.

Our own small hospitals are equipped to make any necessary laboratory tests, including those based on spectophotometry and flame photometry, and service is available around the clock. Each maintains adequate reserves of all standard types of solutions for fluid, electrolyte, protein or blood replacement. There is no question that many physicians can frequently take adequate care of cases of electrolytic imbalance, bleeding or intestinal obstruction by clinical observation combined with intelligent guessing, and the use of only glucose in water and saline solution and blood. However, most patients get along better if the difficult phase of their course is accurately assayed and controlled. An occasional patient is lost without such detailed supervision, regardless of the size of the hospital. In fact, we believe that more lives are lost through minor accidents than through miscarriage of the primary technical procedure. To be sure, if any hospital, large or small, allows surgery to be done that is beyond the capacity of the performer, the anesthetist or the surgical team to handle, catastrophe may be anticipated. Large New England hospitals are often more careful than smaller ones in such respects, but scattered smaller ones are setting standards along these lines of which the most careful of the large might be proud.

The small hospital must be as well equipped and staffed to care for its patient load as the large one is for its greater number of patients, but either succeeds or fails, in terms of the patient himself, largely because of the correctness or incorrectness of care given in the individual case. What solace is found among the living 99 for the wife of the man who dies of a pulmonary embolism ten days after his calf muscles are banged carelessly against a stretcher bar as he is moved from the operating table after a "successful" operation? What has been gained for the patient by a brilliant technical procedure if he is neglected at night and retains pulmonary secretions or inhales regurgitated gastric contents and dies of massive atelectasis? Surgeons as individuals, not the annual census of the institutions in which they practice, control the frequency of these types of carelessness leading to a fatal outcome. Their insistence on vigilance by personnel and administrative provision of properly functioning equipment with which to work, rather than the number of states from which patients were admitted in any

year or the number of names on the hospital payroll, actually determine the outcome for the patient.

Summary

The clinical features of 103 cases of peptic ulcer treated by gastric resection in two small hospitals are outlined. There were 2 deaths. Twelve patients required emergency resection because of massive hemorrhage, and all survived. Principal discussion is devoted to diagnostic and therapeutic measures used in this group of poor-risk, bleeding patients. The problem of gastrointestinal hemorrhage is analyzed, with particular reference to its management in small hospitals.

GASTRECTOMY IN A COMMUNITY HOSPITAL*

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THIS report is an evaluation of the results of gastrectomy in one surgeon's practice in a community hospital (Addison Gilbert Hospital, Gloucester, Massachusetts) since 1946.

Although this procedure is now widely used, and the literature contains many papers on its various aspects,[1-13] no studies regarding its results in community hospitals have been noted. The subject is not static, for mortality and morbidity attend its use and its indications and many technical problems receive wide attention in the published articles.[1-13] In addition, the comparative results of gastrectomy and vagotomy in duodenal ulcer continue to be closely scrutinized.[6,10] It seems highly desirable, therefore, that follow-up studies should be conducted in every hospital where gastrectomy is being done.

At a meeting of Essex South District Medical Society held at the Lynn Hospital in February, 1953, Dr. Francis D. Moore presented some of the findings of the Committee on Surgical Procedures of the National Committee on Peptic Ulcer of the American Gastroenterological Association. Although the results of subtotal gastretomy in duodenal ulcer in the teaching centers were quite uniform (mortality of 2.5 per cent), Dr. Moore had no idea what the results were in the country at large, and he thought the mortality rate might be 10 per cent or higher.

There is a widely held opinion among community surgeons that surgical care in community hospitals and in the centers should not be measured by a double standard. Certainly, one step toward changing such an evaluation is to survey and report results. Colleagues, both in small communities and in the large centers, deserve factual information on surgical activities if these activities are to be judged accurately.

Table 1 outlines the total experience. The average age was fifty-three years, the youngest (a patient with ulcer) being fifteen and the oldest (a patient with cancer) eighty-eight years of age. These patients were unselected and represent all cases in which gastrectomy was considered necessary, regardless of risk. During the same period only 3 gastroenterostomies were performed — all for scar stenosis of the pylorus resulting in complete obstruction.

Duodenal Ulcer

Of 41 patients with duodenal ulcer 35 were males and 6 females, the average ages being forty-six and fifty-one years respectively. The ages ranged from fifteen to seventy-five years. The youngest, a fifteen-year-old boy, had a history of difficulty since childhood. The findings were complete obstruction of the pylorus with a large (2 by 4 cm.) ulcer of the posterior wall penetrating deeply into the pancreas. This patient has been followed for eighty-one months and has remained symptom free and in good health (he went through college on an athletic scholarship).

The average duration of symptoms in the patients with duodenal ulcer was ten years. Indications were intractability in 14 cases, repeated hemorrhage in 8, obstruction in 10, previous gastric surgery and continued pain in 4, gallstones and ulcer pain in 3 and, finally, a question of cancer in 3. The last group represented patients whose ulcers appeared, on x-ray examination, to be situated in the prepyloric area.

Gastric Ulcer

The gastric-ulcer group comprised 15 patients, 8 men and 7 women, with average ages of fifty-five and sixty-two years respectively. The age range was from thirty-five to seventy-three years. The average duration of symptoms was five years. Indications were intractability in 12 cases, hemorrhage in 5 and a question of cancer in all 15. The degree of suspicion of cancer varied in these cases, but in none was the suspicion great. However, a patient in the cancer group was operated on for gastric ulcer, and even at time of operation the ulcer excited no more suspicion of malignant regeneration than that of the average patient in the benign group.

Cancer

This group of patients demonstrated the well known gloomy outlook. Of the 9, resection was performed for palliation in 4 and for cure in 5. During the same period, 7 other patients were explored and

*Presented at the annual meeting of the New England Surgical Society, Watch Hill, Rhode Island, September 12, 1953.
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found to present conditions that offered no hope for palliation. There were 6 men and 3 women, with an average age of sixty-two and seventy-seven years respectively. The ages ranged from thirty-seven to eighty-eight years. Two of the 9 patients underwent total gastrectomy, their ages being thirty-seven and seventy-eight years. The younger died of recurrence in five months. The seventy-eight-year-old patient was well eighteen months after operation. This patient is of interest in that he entered the hospital with complete pyloric obstruction. At operation he was found to have, in addition to the prepyloric tumor causing obstruction, a huge carcinoma of the fundus.

Massive Hemorrhage

The 7 cases in this group represented patients operated on during massive uncontrollable bleeding. There were 6 men and 1 woman, the ages averaging sixty-five years. The oldest patient was eighty-five years. In all these patients blood was rapidly replaced after admission, and surgery was resorted to when the condition of the patient and laboratory studies indicated rapid continued bleeding. An average of 6 units of blood was used before operation, and 2.5 units during and 2.5 units after operation.

The sources of bleeding were gastric ulcer in 2 patients, duodenal ulcer in 2, jejunal ulcer in 2 and gastritis in 1. In all except the last patient a large artery was actively bleeding at operation.

In 1 case, a forty-six-year-old man, who had had a perforated ulcer at twenty years of age and a posterior gastroenterostomy two years later and who had subsequently had repeated episodes of ulcer pain, entered the hospital after a series of tarry stools. Blood replacement was being carried out in the usual manner, when he had a sudden recurrence of rapid bleeding and became unconscious without pulse or blood pressure. Two simultaneous rapid intravenous transfusions were given without effect. No anesthesia was required to open the radial artery, and no bleeding appeared from it when a cannula was inserted. Intra-arterial transfusion was given, and after 200 cc. of blood the patient responded. A systolic blood pressure of 70 was maintained. He successfully underwent subtotal gastrectomy and jejunal anastomosis. In this patient, who was a fisherman with peripheral vascular disease, necrosis of the hand later developed.

Table 1. *Summary of All Cases of Gastrectomy, 1946–1953.*

Indication for Operation	No. of Cases
Duodenal ulcer	41
Gastric ulcer	15
Massive hemorrhage*	7
Cancer	9
Other disease	3
Previous gastric surgery†	10

*Patients operated on during uncontrolled massive hemorrhage.

†Operations included 5 for perforated ulcers, 4 gastroenterostomies and 1 partial gastrectomy 17 yr. earlier.

The small group of 3 cases, listed under "other diseases" in Table 1, comprises 3 patients who were operated on for gallstones. In addition to cholelithiasis, prepyloric tumors were found. Two of these tumors proved histologically to be aberrant pancreatic tissue, and the third was hypertrophy of gastric muscle. The last patient has continued to have symptoms of "bilious vomiting," present for twenty years before operation, in spite of revision of the stoma a year later in another city to which she had moved. The other 2 patients have been well.

Risks

An estimation of risk was made, and 50 patients were considered to be good, 10 fair, and 15 poor risks. There were 11 patients seventy years of age or over, 9 who had severe cardiorenal disease and 7 who were very obese.

Table 2 indicates the lesions found at operation in the 56 patients with duodenal or gastric ulcer. It will be noted that in 49 real pathological findings were encountered.

Table 2. *Lesions Encountered at Operation in 56 Patients.**

Indication for Operation	Patients with Scar	Patients with Ulcer	Patients with Ulcer with Penetration	Patients with Obstruction
Gastric ulcer	0	8	7	0
Duodenal ulcer	7	12	14	8
Totals	7	20	21	8

*Not including patients with massive hemorrhage.

Management

Anesthesia

Continuous spinal and endotracheal anesthesia with oxygen and ether were used in an approximately equal number of patients.

Operation

Table 3 indicates the types of procedure. In all cases, more than 70 per cent of the stomach and the pylorus were removed. In the Billroth I and Hofmeister procedures all the lesser curvature was included in the resection. In the Polya operations nearly all the lesser curvature was removed. In all cases a short loop of jejunum was used for anastomosis, with the distal loop at the greater curvature. The choice of procedure depended on the situation at hand, large size of omentum and omental adhesions in the lower abdomen usually indicating posterior colic anastomosis.

In all but 2 patients with duodenal ulcer, the lesion was removed. Jejunostomy drainage was used in 6 cases. Additional procedures included

jejunojejunal anastomoses in patients who had had previous gastroenterostomies and removal of the greater omentum, spleen and gall bladder. Simple Penrose drains were used when indicated but were never placed directly to a suture line. In an appreciable number of the large ulcers, both duodenal and gastric, that penetrated deeply into the pancreas, copious serous drainage occurred, accompanied, however, by a smooth convalescence in all cases. The greater omentum was removed as well as the mesentery in the 7 most recent cases of gastric ulcer and all patients with gastric cancer operated on for cure.

Inside catgut suture and outside silk on the anastomosis were routine. In wound closure stainless-steel wire suture was most widely used.

An average of 2 units of blood was given during and after operation. All patients were kept on naso-gastric suction until peristalsis became evident. The great majority were ambulatory on the day after surgery. In all cases the lower legs were kept bandaged for five days after operation.

Table 4 gives the immediate results in 75 patients undergoing gastrectomy. The nonfatal complications included mild pulmonary atelectasis, urinary retention and cystitis, uremia, 1 wound infection and 1 case of necrosis of the hand from intra-arterial transfusion. In 1 case empyema of the gall bladder developed from a stone of millet-seed size impacted in the cystic duct.

Early postprandial distress, frequently reported, was rarely noted and was not of sufficient account to justify comment. This group included 3 patients with early and transient diarrhea and 3 with occasional regurgitation of bile for several weeks. Two patients had mild occasional dumping syndrome, which in 1 patient has disappeared entirely and in the other is represented by a full feeling that is no longer annoying and occurs less than once a month.

There were 4 postoperative hospital deaths, in 3 of which post-mortem examination was performed.

Deaths

The first patient was a fifty-six-year-old man who was operated on during uncontrollable massive hemorrhage. He had had duodenal ulcer for years as reported by his physician and radiologist. At operation, hypertrophic gastritis was found, and subtotal gastrectomy was done, with a good immediate response. Six days after operation, paroxysmal tachycardia, recurrent hemorrhage and cardiac decompensation occurred and were followed by death. At autopsy no bleeding point was found, only gastritis and normal postoperative surgical findings being apparent.

Another patient, an eighty-eight-year-old woman, had constant pain and vomiting caused by pyloric obstruction from carcinoma. She lived for ten days after resection, dying in coma. There was no evidence of surgical difficulty. Permission for autopsy was not obtained. The cause of death was cerebral thrombosis.

In a seventy-year-old obese man, complete pyloric obstruction followed a penetrating duodenal ulcer. After operation atelectasis and leakage from the duodenal stump developed; drainage was performed, and he recovered from this procedure and was ambulatory, with normal gastrointestinal function. A pulmonary embolism occurred 28 days after operation. Autopsy demonstrated the pulmonary

TABLE 3. *Types of Procedure.*

PROCEDURE	PATIENTS WITH DUODENAL ULCER	PATIENTS WITH GASTRIC ULCER	PATIENTS WITH MASSIVE HEMORRHAGE	PATIENTS WITH CANCER	PATIENTS WITH OTHER DISEASES	TOTALS
Hofmeister anterior colic resection	17	1	1	2		21
Hofmeister posterior colic resection	7	2	0	0		9
Polya anterior colic resection	9	0	3	1	1	14
Polya posterior colic resection	8	3	2	1	2	16
Billroth I resection	0	9	0	3	0	12
Total gastrectomy	0	0	0	2	0	2
Additional procedure	8	9	3	5	3	28
Drain	17	9	4	4	3	37

TABLE 4. *Immediate Results in 75 Patients.*

AVERAGE POSTOPERATIVE HOSPITAL STAY	AVERAGE DURATION OF POSTOPERATIVE GASTRIC SUCTION	AVERAGE DEGREE OF POSTOPERATIVE TEMPERATURE RISE	NO. OF PATIENTS WITH ELEVATED TEMPERATURE* FOR 2 DAYS	NO. OF PATIENTS WITH NONFATAL COMPLICATIONS	NO. OF DEATHS	NO. OF AUTOPSIES
days	*days*	*°F.*				
15.7	2.8	1.6	7	11	4	3

*TO 100.6°F.

embolus as well as local inflammation about the stump.

The last of these patients was a sixty-four-year-old man with a high, fixed nonprotein nitrogen (77 mg. per 100 cc.) In addition to coronary sclerosis he had had a duodenal ulcer with obstruction and penetration into the gall bladder. In another alcoholic patient serious hepatitis has developed.

Four patients have had surgical procedures since gastrectomy; 1 for small-bowel obstruction in the pelvis from a previous gynecologic operation, and another for bowel obstruction from an adhesion just distal to the stoma. A patient with empyema of the gall bladder required drainage. The fourth patient was recently operated on for gangrenous appendix.

Patients were requested to state their opinion of the results of their surgical treatment and indicate whether well satisfied, satisfied or dissatisfied. It will be noted that none were dissatisfied, and 53 were well satisfied.

Table 6 gives the follow-up data of the patients operated on for gastric cancer.

Table 7 shows results of the Billroth I procedure in 12 patients operated on for gastric lesions. Seven of the 12 patients had radical subtotal gastrectomies including the greater omentum and the mesenteries, as well as the first portion of the duodenum.

The gastric stump was prepared by the Hofmeister method; the Furniss clamp was used along the lesser curvature, all of which was removed. In all cases as much stomach was resected as if a Hofmeister modification of a Billroth II procedure had been used. This operation requires the presence of a normal duodenum. The average time required for operation in the entire group of 75 patients was a hundred and sixty-one minutes. In the 12 patients undergoing Billroth I gastrectomy, the average time required was a hundred and fifteen minutes.

TABLE 5. *Follow-up Data in 59 Patients with Ulcer.*

DATUM	NO. OF CASES
Symptoms:	
Ulcer pain	0
Hemorrhage	1*
Gastrointestinal symptoms	1†
Alcoholism	6
Hernia	2
Weight‡:	
Loss of average of 18 pounds	32
Gain of average of 13 pounds	15
No change	10
Subjective result:	
Working activity not limited	57
Patient well satisfied	53
Patient satisfied	6
Evaluation:	
Excellent	45
Good	12
Fair	2

*Silent hemorrhage, in an alcoholic patient with a negative x-ray examination.

†Alcoholic patient.

‡The average loss for the entire group was 6 pounds.

prepyloric ulcer, a question of cancer and uncontrollable pain and vomiting. After resection he lived for twenty-eight days, dying of uremia. At autopsy chronic nephritis, pleuritis, pericarditis and normal surgical findings were observed.

FOLLOW-UP DATA

Table 5 gives the follow-up results of the 59 surviving patients who were operated on for gastric and duodenal ulcer, including those with massive hemorrhage. (One patient of the 6 who survived massive hemorrhage subsequently died of cancer.)

TABLE 6. *Follow-up Data in 9 Patients with Cancer.*

DATUM	NO. OF CASES	
Death from recurrence*	3	Average 8 months
Death from other causes	2	
Postoperative death	1	
Patient living and well	3†	

†One (65 yr. of age) for 12 mo., in excellent health, with 10-lb. weight gain; another (78 yr. old) for 18 mo. after total gastrectomy, well, with weight gain; and 1 (80 yr. of age) for 34 mo., well except for slight cerebral accident.

TABLE 7. *Results of Billroth I Procedure in 12 Patients with Gastric Lesions.**

OPERATION FOR GASTRIC ULCER	OPERATION FOR GASTRIC CANCER	RADICAL RESECTION	AVERAGE POST-OPERATIVE HOSPITAL STAY	AVERAGE TEMPERATURE RISE	MORBIDITY	AVERAGE WEIGHT GAIN	PATIENT'S OPINION OF RESULT	RESULT EVALUATED EXCELLENT	RESULT EVALUATED GOOD
NO. OF CASES	NO. OF CASES	NO. OF CASES	*days*	*°F.*		*lb.*		NO. OF CASES	NO. OF CASES
9	3	7	14	1.4	0	5	Well satisfied	11	1

*Average age 60 yr.; oldest patient 80 yr. of age.

The follow-up study ranged from four to eighty-one months. The average was twenty-eight and seven-tenths, and the mean twenty-three months.

Alcoholism has been an occasional problem in that patients with alcoholic tendencies operated on for severe ulcer have subsequently been able to drink alcoholic beverages without real difficulty. One patient, a profound alcoholic, has recurrent diarrhea and vomiting; x-ray studies remain completely negative. Surgery was mandatory, for she

Summary

The results of gastrectomy since 1946 in 75 patients in a community hospital are reported. The average follow-up period was twenty-eight and seven-tenths months. There were 4 hospital deaths, with autopsies in 3. At the time of this report the results in 57 of the 59 patients with ulcer are considered to be good or excellent, and those in 2 are fair. The patients' own estimates indicate that 53 are well satisfied, 6 satisfied, and none dissatisfied.

In 12 patients with gastric lesions, a Billroth I procedure required a shorter operative time and yielded excellent results.

The survey of this small group of cases shows results reasonably similar to those reported from the large medical centers.

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MUCOSAL HYPERTROPHY OF THE STOMACH*

Report of a Case

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BOSTON AND WORCESTER, MASSACHUSETTS

THE clinician and the roentgenologist are confronted periodically with the management of a patient in whom large gastric rugal folds have been found. The differential diagnosis must include mucosal spread of an anaplastic carcinoma, lymphoma, hypertrophic gastritis and mucosal hypertrophy, with the possible inclusion of a chronic inflammatory granuloma, such as sarcoid or syphilis.

Mucosal hypertrophy of the stomach has been variously termed Menetrier's disease[1] (*polyadénome en nappe*) and tumoral hypertrophy,[2] and is often mistakenly called chronic hypertrophic gastritis and diffuse gastric polyposis.[3] It is uncommon, and its natural history unknown. The changes that take place in the gastric mucosa have no anatomic similarity to chronic hypertrophic gastritis or gastric polyposis, nor is there any reason to believe that the condition is a forerunner to malignant change. Mucosal hypertrophy is characterized by true thickening of the gastric mucosa, resulting from elongation of the otherwise normal glands. The giant rugae thus formed were likened to cerebral convolutions in Menetrier's description of the disease in 1888. The mucosal thickening cannot be ascribed to inflammatory infiltration and edema, for there is no increase in the usual complement of interstitial cells within the membrane.

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Case Report§

A 58-year-old candy salesman entered the Worcester Memorial Hospital with the diagnosis of a "stomach tumor." Six months previously nausea and vomiting had first developed, either during a meal or immediately afterward, particularly when he had eaten quickly. Vomiting relieved the nausea. There was no associated dysphagia, hematemesis, epigastric distress, melena, change in bowel habits or weight loss. One month before admission, during a routine annual checkup at another hospital, a "tumor" of the stomach was noted on x-ray study, and gastroscopic examination showed hypertrophied gastric rugae without mucosal ulceration. A biopsy was reported as "acute gastritis." The gastric aspirate was stated to contain no free hydrochloric acid, and only "a few units" of combined hydrochloric acid. A Papanicolaou smear of the gastric aspirate was reported as negative.

Physical examination revealed a healthy-appearing and well nourished man. The only positive physical finding was a large, nontender, movable mass in the left upper quadrant of the abdomen. The blood pressure was 150/75.

The temperature, pulse and respirations were normal.

Urinalysis was negative. The hemoglobin was 12.9 gm., and the white-cell count 3550, with 78 lymphocytes, 20 neutrophils and 2 eosinophils. The total serum protein was 5.34 gm. per 100 cc., with an albumin of 4.3 and a globulin of 1.04 gm. per 100 cc. The fasting blood sugar and nonprotein nitrogen were normal. A guaiac test on the stools was negative. A gastrointestinal series suggested greatly enlarged rugal folds, with filling defects in the upper third of the stomach (Fig. 1). Although repeat cytologic examination of the gastric aspirate was negative for tumor cells, it was believed that a malignant tumor could not be ruled out without operation.

Through a thoracoabdominal approach total gastrectomy and splenectomy were performed, followed by an anastomosis between the esophagus and duodenum. Recovery was uneventful.

The stomach was very large, measuring 17.5 cm. along the lesser and 48 cm. along the greater curvature. Externally, it was not abnormal apart from dilatation of the veins along the lesser

§We are indebted to Paul Ware, M.D., for permission to publish his case.

curvature. The rugal folds throughout the cardia, fundus and body of the stomach were greatly hypertrophied, pale pink and glistening. The folds ran longitudinally to the prepyloric area,

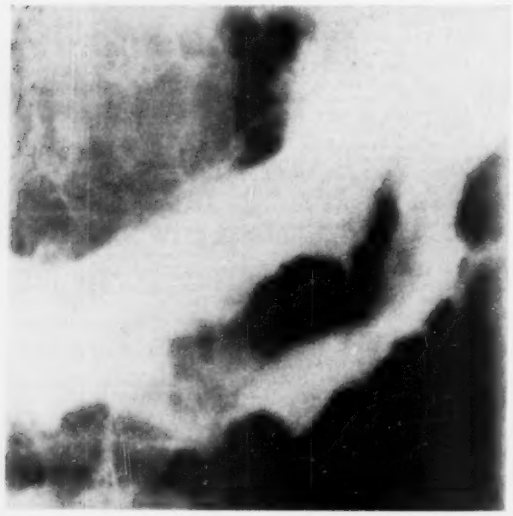

FIGURE 1. *Detail View of the Barium-Filled Stomach at the Level of the Gastric Angle, Showing Greatly Thickened Rugal Folds.*

where the mucosa became flat and grossly normal. The large convolutions in the fresh state averaged 2 cm. in width and 2 cm. in height (Fig. 2). The musculature averaged 3 mm. in

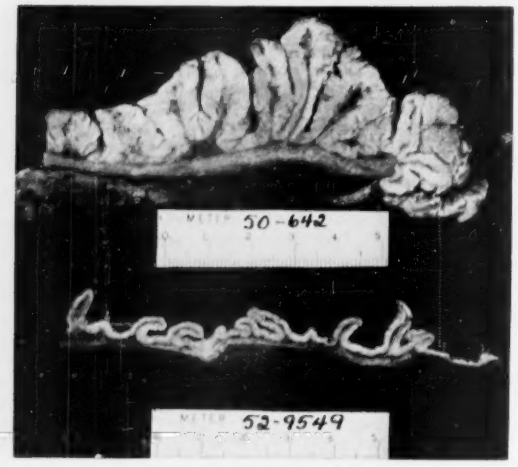

FIGURE 2. *Cross-Sectional Comparison of a Normal Stomach (below) with the Stomach in Giant Mucosal Hypertrophy (Fixed Specimens).*

thickness. The serosa appeared normal. The regional lymph nodes were not enlarged, and the spleen was not remarkable.

Microscopically, the gastric glands were greatly increased in height but were not tortuous and had a normal configuration. The individual cells appeared normal. In some areas small cysts had formed, usually at the base of the elongated glands but occasionally in the mid-zone (Fig. 3). Mitotic activity in the neck zone of the glands was not excessive. In a few areas herniation of the glands through the muscularis mucosae had

FIGURE 3. *Low-Power Photomicrograph, Showing the Extreme Elongation of the Normally Configurated Mucosal Glands except for Occasional Cyst Formation.*
There is no increase in inflammatory cells.

occurred. Intestinal metaplasia of the gastric epithelium was not found.

DISCUSSION

The practical clinical importance of mucosal hypertrophy of the stomach lies in its recognition

FIGURE 4. *Cross Section of the Stomach in a Case of Multiple Polyposis (Fixed Specimen).*
The patient, a fifty-eight-year-old man had had mild epigastric fullness and distress for two months. He was well fifteen years after operation. Note the individual polyps.

and differentiation from hypertrophic gastritis, malignant tumor and diffuse polyposis (Fig. 4). The last condition is characterized by clusters of

closely placed polyps, either sessile or pedunculated, but all with separate mucosal attachments. Microscopically, the glands are elongated and tortuous, and the cells may be atypical. Dolan and Sherman[2] recently reviewed the subject of mucosal hypertrophy and collected 26 cases, including the one they reported. With justification, they stressed the necessity of distinguishing mucosal hypertrophy from chronic hypertrophic gastritis.

In a review of the reported cases it is obvious that the symptoms of this gastric change are not impressive. Often, there is a history of epigastric or upper abdominal discomfort without a relation to food. Ingestion may relieve or aggravate the discomfort. In addition, there may be anorexia, eructation, nausea and vomiting. Some patients have lost weight, have been anemic and have complained of lethargy, headaches, constipation or diarrhea. There are no pertinent physical findings.

The roentgenologist is faced with a difficult problem, since mucosal hypertrophy may mimic infiltrating carcinoma and lymphoma; furthermore, the giant folds cannot be obliterated by pressure on the stomach. The gastroscopist can visualize the large rugae that show no ulceration, and here again, the folds do not recede with inflation of the stomach. Still one cannot be certain that malignant infiltration is not present. A biopsy through the gastroscope is encouraging in a negative sense.

Helpful laboratory findings have not been recorded. In the gastric juice the free hydrochloric acid may be normal or diminished; according to Dolan and Sherman[2] there is a tendency to hypochlorhydria. Blood in the gastric aspirate and stool is unusual. Balfour et al.[4] have discussed the occurrence of hypoproteinemia in association with giant hypertrophy of the gastric rugae. Their patient suffered from severe hypoproteinemia, which was not relieved by medical measures but was restored to normal after total gastrectomy.

The finding of a neutropenia with a relative lymphocytosis in the case reported above may have been an error, since the determinations were not repeated. This observation has not been recorded in the condition by others.

From the standpoint of anatomy this case appears to differ in no way from that usually described. Typically, the stomach shows an unaltered serosa, apart from dilatation of the blood vessels. The wall is not stiffened, and the mucosa in a large area, or diffusely, exhibits enormous, hyperemic, freely movable rugae like cerebral convolutions that are often covered by an excessive mucous secretion. Microscopically, there is elongation of the gastric glands, with retention of the normal pattern and cellular detail. There may be marked intestinal metaplasia, microscopic cyst formation and herniation of the glands through the muscularis mucosae. There is no increase in the inflammatory-cell population of the mucosa. There is no real indication that the condition precedes cancer, although Matzner, Raab and Spear[5] reported a case of anaplastic cancer developing ten years after the discovery of benign giant gastric rugae in a fifty-year-old man.

Summary and Conclusions

A case of mucosal hypertrophy of the stomach, a rare but distinct condition characterized by the formation of giant rugal folds made up of elongated glands of normal configuration and differentiation, is reported. There is no apparent relation to gastritis, and peptic ulceration does not occur. Its practical importance lies in the necessity for differentiation from anaplastic carcinoma, lymphoma and multiple polyposis, which may produce the same roentgenologic and gastroscopic appearance. Hypoproteinemia and achlorhydria may occur. There is no evidence that this is a precancerous condition.

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MEDICAL PROGRESS

HYPERTENSION IN PREGNANCY

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IN discussions of hypertension in pregnancy the term "toxemias of pregnancy" has been discarded. Pre-eclamptic toxemia is the only potentially toxic syndrome, and even that has not been established. Hypertensive disease in pregnancy is classified in three groups, as follows: essential hypertension (patients with pre-existing vascular disease); renal disease (patients with pre-existing renal disease, with or without hypertension); and pre-eclamptic toxemia and eclampsia. It is believed that a superimposed pre-eclamptic toxemia is likely to occur in the first two groups. It is difficult to prove the development of a superimposed lesion on a pre-existing one, but because the early effects of pre-eclamptic toxemia are primarily renal and vascular, it is only reasonable to assume that a previously damaged renal or vascular system predisposes to the development of pre-eclampsia. However, it may take months or years of careful follow-up study to make a definite diagnosis and it is often impossible, clinically, to classify individual cases.

To understand properly the problem of hypertensive disease in pregnancy, one must first comprehend certain physiologic alterations in the normal pregnant woman. The most important of these in relation to hypertension is the effect of pregnancy on the water and electrolyte metabolism. Dieckmann[1] has shown that there is a delayed excretion of water and sodium in the normal pregnant woman. Why this is so is not clearly understood. The possibility of the presence of the antidiuretic hormone of the pituitary gland in abnormal amounts or a change in adrenal physiology particularly involving desoxycorticosterone has been considered. However, the basic clinical fact that pregnant women store water and sodium is well established.

About 60 per cent of all pregnant women have edema of some type. The dependent edema of the legs is largely mechanical in origin owing to increased venous pressure. This is a common clinical finding and is of no particular importance in relation to the development of hypertension. Generalized edema of the face, hands and abdominal wall is of much more significance. This is not mechanical in origin and definitely indicates fluid retention. The most serious form of water and sodium retention is the fluid held in the extravascular spaces of the various tissues and organs. Normally about 35 per cent of the body fluid is stored in these spaces, and much more can be stored there. In addition, there may be a marked increase in intracellular fluid and displacement of cellular potassium by sodium. This tissue storage of fluid is most significant in relation to the development of pre-eclampsia. Frequently, there is no visible edema even when large amounts of fluid are retained. Rapid and excessive weight gain is the only clinical evidence of the retention of this fluid. Rapid and excessive weight gain in the pregnant woman is primarily fluid retention.

The fluid retention is due to sodium retention. Lack of proper sodium excretion by the kidneys and excess sodium intake in the diet are the primary causes. It is well to remember that the sodium intake in the diet of the average woman is considerably in excess of what she can efficiently eliminate in the pregnant state.

Another important physiologic and clinical disturbance in pregnancy is the increased load on renal function. Pregnancy is about the best clinical test of kidney reserve. There are many women who have sufficient kidney function for the normal nonpregnant state but whose kidney reserve has been damaged by previous renal disease. In the nonpregnant state, no tests are sufficiently delicate to demonstrate the kidney lesion. Yet with the added load of pregnancy definite signs of renal failure may well develop. Just what causes the added load and strain in the kidney in pregnancy is not well understood. Changes in adrenal function, the excretion of large amounts of steroids, various metabolic changes of pregnancy, increased blood volume and cardiac work, and fetal metabolism may be involved. The fact remains that a normal pregnancy may be too great a load for a kidney with a low reserve.

RENAL DISEASE

Renal disease in pregnancy is one of the most serious complications and one that offers great difficulty in diagnosis and treatment. With the definite presence of this disease, the prognosis for both mother and baby is much worse than in either essential hypertension or pre-eclamptic toxemia. Severe chronic nephritis causes lowered fertility. One does not see many such patients who have become pregnant. A more frequent problem is the early chronic nephritis with kidneys that still function reasonably well in the nonpregnant state.

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From a clinical point of view, it is frequently impossible to make a differential diagnosis between different types of chronic renal damage. The most common type appears to be chronic pyelonephritis; many patients with this form of disease have had acute pyelonephritis in childhood, with residual renal damage and lowering of kidney reserve. Frequently, the diagnosis was not established at the time of the acute episode. Therefore, there may be no past history of the disease (although a past history is of great value, its absence by no means rules out the condition). Patients may have a sufficient kidney reserve to enable them to live in good health in the nonpregnant state. The addition of the load of pregnancy may bring on the first signs and symptoms. If a patient is seen first in the last trimester of pregnancy, the differentiation from pre-eclamptic toxemia may be most difficult. However, with repeated pregnancies, signs of renal damage tend to appear earlier and become more severe. This appearance in repeated pregnancies with increasing severity is in direct contrast to pre-eclampsia, which does not tend to be present in successive pregnancies or become successively more severe.

Certain specific problems are more acute in renal disease than in other hypertensive states. The first is that each pregnancy causes further injury to an already damaged kidney. The life expectancy of the patient is shortened by each pregnancy. Signs of renal failure appear earlier and become more acute. Therefore the first pregnancy of such a woman offers the best chance for a healthy living child; with each succeeding pregnancy, the prognosis for child and mother becomes poorer. Every effort should be aimed at a successful conclusion to the first attempt.

In addition to the danger to the mother, the established diagnosis of chronic renal disease offers a less than 50 per cent prognosis for survival of the child. The fetus frequently dies in utero during the last month, or else the infant does not survive labor or does not have sufficient vitality to survive after birth. Although the placenta does not tend to separate in chronic nephritis as it does in acute pre-eclampsia, there is a marked tendency to infarction of the placenta. Why this is so is not understood, but the typical placenta in chronic nephritis is small and contains many white and yellow infarcts. This is in contrast with the placenta of pre-eclampsia, in which acute hemorrhagic (dark-red) infarcts tend to form. The infarcts in chronic nephritis are a gradual rather than an acute formation. Frequently, a third of the placenta is infarcted; this is the limit of safety for the baby's survival. In cases of death in utero, half or more of the placenta may be infarcted; because of lack of proper nutrition, the fetus tends to be small for its stage of uterine maturity.

The diagnosis of the milder forms of chronic nephritis offers great difficulty. Any patient who shows albumin or casts in the urine during the first half of pregnancy must be considered as a possible case of chronic nephritis. These patients may or may not have an accompanying hypertension. Renal-function tests should be done, particularly to demonstrate the patient's ability to concentrate the urine; a fixed or limited specific gravity is definite proof of this condition and gives a poor prognosis. Other tests are of little value in milder cases. A rise in nonprotein nitrogen or other changes in blood chemistry are rarely found in the mild case. An established hypertension, in addition to the urinary findings, makes the prognosis much worse. The established severe nephritis, with changes in renal-function tests, signs of early renal failure and hypertension, means a very poor prognosis. Eye-ground changes may be of diagnostic importance.

The treatment of chronic nephritis in pregnancy consists primarily in avoidance of excessive weight gain and of fluid and electrolyte retention and in sufficient rest. A high-protein diet is indicated to avoid hypoproteinemia from loss of albumin and the accompanying development of edema. A low-sodium intake is probably the most important factor in diet. With already damaged kidneys, the retention of sodium and water becomes a greater problem than in the normal pregnant woman. Any excessive retention of fluid tends to precipitate renal failure. Therefore, weight gain must be kept at a minimum.

It is most important that the patient be carefully watched and examined at frequent intervals. A few days or a week may bring about a serious change in her condition. Edema or weight gain, increase in albuminuria and hypertension are all signs of impending trouble. Visual disturbances and headaches are of prognostic significance. As long as there is no change in the general clinical picture the patient may be safely carried on until the fetus becomes sufficiently mature for survival.

In many cases, usually in the last trimester, sudden signs of renal failure develop with fluid retention and increase in weight, followed by increase in albumin and casts in the urine, occasionally with red blood cells in the sediment and a rise in hypertension, especially affecting the diastolic pressure. There may also be a rise in nonprotein nitrogen, indicating the development of a pre-eclamptic toxemia superimposed on an already damaged kidney (most authorities believe this to be so although it is difficult to prove). Certainly, a developing toxemia is more rapid and virulent in the presence of renal disease than in a patient with normal kidneys. The combination of toxemia with renal failure is a most critical state. At the earliest signs of renal failure, the pregnancy must be terminated for the sake of both the mother and the

baby; the former may go into irreversible renal failure, and the latter may die in utero.

In the patient whose status is unchanged up to the last month, termination of pregnancy must be seriously considered. Even if there is no sign of impending renal failure, there are definite indications for delivery. The last month of pregnancy may cause serious and permanent damage to the kidney, since this is the time when renal failure is most likely to occur. The sooner the load is taken off the kidney, the less chance of serious damage. It is also during the last month that the fetus is most likely to die in utero. Increasing placental infarction, in addition to any toxemia, may have a serious effect on the baby. If it does not die in utero its vitality may be so lowered as to make it unable to survive birth. The whole situation must be carefully considered, and the optimum time for delivery chosen.

The method of delivery depends on the individual case. In a multipara with the vertex engaged and a soft and partly dilated cervix, induction of labor by rupture of the membranes may be the procedure of choice. In most primiparas cesarean section is indicated. In the critically ill patient, one must weigh the risk of abdominal surgery and accompanying shock against the frequent difficulty of induction from below.

The patient with very mild nephritis and no signs of toxemia or increasing renal or vascular damage during the pregnancy and with no hypertension may be safely allowed to go to term and be delivered by spontaneous labor.

Essential Hypertension

Essential hypertension is believed to be primarily due to vascular disease involving spasm of the arterioles. Of unknown etiology, it resembles pre-eclamptic toxemia in many ways and yet is a distinct entity. In the early stages no kidney damage can be demonstrated, and its presence in pregnancy is a much less severe complication than that of renal disease. In its advanced stages when kidney damage can be demonstrated, it responds to pregnancy as any other type of renal disease.

Frequently, pregnancy is the precipitating factor in latent and potential hypertension, occurring during the first pregnancy in many young women with no previous history of hypertension. Once this condition has developed, it tends to remain after termination of the pregnancy. In a patient with essential hypertension appearing for the first time during pregnancy pre-eclamptic toxemia may be thought to be developing, but in these early cases there rarely is albuminuria or signs of kidney damage.

Other young women have a pre-existent essential hypertension that is apparent before the onset of pregnancy; if the condition is mild, the prognosis for the pregnancy is good. The few patients who have malignant hypertension before pregnancy constitute a dangerous and critical group.

The clinical diagnosis of essential hypertension in pregnancy is made from the pre-existence or the appearance of hypertension early in pregnancy. With negative urinary findings and no signs of kidney disease, the diagnosis is made by exclusion.

The classification is important because a patient with mild essential hypertension has a much better prognosis than one with chronic renal disease. Although all authorities do not agree, it is my opinion that essential hypertension is unaffected by pregnancy unless a superimposed pre-eclamptic toxemia develops. With an already damaged vascular system and arteriolar spasm and possible arteriolar constriction, the patient already has the vascular picture of pre-eclampsia. Therefore, it is clear (and this is substantiated clinically) that these patients have a greater tendency to pre-eclamptic toxemia than those with a normal vascular system. However, most of these patients, under proper care, pass through pregnancy — and many through several pregnancies — without any temporary or permanent change in the hypertension.

This by no means indicates that this group should be treated casually; they must be under strict care and supervision. Any rise in blood pressure or development of albuminuria warns of impending pre-eclampsia. This does not usually occur until the last trimester. Such signs should be regarded with the utmost seriousness because if pre-eclampsia develops the toxemia is likely to be more severe and rapid than that in the patient with a normal vascular system. Termination of pregnancy is indicated as soon as true pre-eclampsia develops.

The treatment of essential hypertension in pregnancy is to direct all endeavor to the prevention of the development of pre-eclamptic toxemia. This, again, is largely dependent on the prevention of water and electrolyte retention. The patient should be on an extremely low-sodium intake. This diet must be carefully explained and given to the patient on a printed sheet. She must be warned of the danger of her condition, and it must be explained that any departure from her instructions may cause serious trouble. Her weight must be carefully checked at least once a week during the second half of pregnancy. The development of any edema or excessive weight gain (2 pounds a week or 5 pounds a month) must be regarded as fluid retention, and the patient as being in the earliest stage of pre-eclampsia. Hospitalization is the treatment of choice, although in exceptional circumstances the patient may be treated at home under close supervision for a week. A serious attempt must be made to produce a diuresis. All possible sodium must be eliminated; ammonium chloride may be given, and the patient kept at complete bed rest. It is at this stage of fluid retention that pre-eclamptic toxemia may be prevented. Once albuminuria and an increase in hypertension

have occurred an irreversible stage of the disease has developed. With clinical signs of pre-eclampsia, either mild or severe, the disease will be present until the termination of pregnancy.

If on treatment at home or in the hospital a marked diuresis has been accomplished within a week, the prognosis remains fairly good. A good response to dehydration consists of a weight loss of 8 to 15 pounds. A check of fluid intake and output is of help but is difficult especially in hot weather, when fluid loss through the skin and lungs may be excessive. Weight loss is the best and simplest guide to the patient's progress.

The question of free or restricted fluids during the period of diuresis has been much debated. The conclusion appears to be that the elimination of sodium is the important factor and that possibly free fluid aids in flushing out the sodium.

For the patient who does not respond to attempts at diuresis by losing weight or even continues to gain, hospitalization is necessary. Albumin shortly appears in the urine, and the blood pressure begins to rise; the patient has reached the stage of true pre-eclampsia and must be treated as such. With the progress of the disease, termination of pregnancy is soon indicated. As in chronic nephritis, the type of delivery depends on the individual case. Those favorable for induction and delivery from below should be treated in this manner. In those unfavorable for this procedure, a cesarean section may be the method of choice.

Women who have passed uneventfully through a pregnancy complicated by essential hypertension are usually unharmed by the pregnancy. In these cases, further pregnancies are relatively safe unless there has been some exacerbation of the disease. On the other hand, patients with the more severe types, especially those complicated by pre-eclampsia, should be warned against frequent and repeated pregnancies.

Pre-eclamptic Toxemia

Pre-eclamptic toxemia is a hypertensive state appearing usually in the last trimester and disappearing shortly after the termination of pregnancy. As mentioned above, it may be superimposed on previously existing renal vascular damage. This disease is most common in young primiparas although it also appears in older multiparas. Pre-eclampsia has no great tendency to reappear in subsequent pregnancies. The patient who has recurring hypertension with increasing severity in succeeding pregnancies probably has some underlying renal or vascular disease.

Certain abnormalities of the pregnancy state tend to increase the incidence of pre-eclampsia. Among these are twin pregnancies, hydramnios, overlarge babies and hydatidiform mole. These conditions have in common an overdistention of the uterus.

The etiology of pre-eclampsia is unknown. Many theories have been propounded, but they have not stood the test of time. The most commonly accepted assumption at present is that because of some interference with the uterine circulation, anoxemia of relative severity develops in the placental area. Owing to this anoxemia there is a breakdown of certain toxic substances that appear in the maternal circulation. However, no such toxic products have been isolated. Many investigators have tried, without success, to demonstrate a vasopressor or antidiuretic substance in the blood of pre-eclamptic women. Hofbauer[2] first suggested that the secretion of the posterior lobe of the pituitary gland might be the cause of pre-eclampsia. Dieckmann[1] states that patients with pre-eclampsia show a rise in blood pressure after injections of Pituitrin; this does not take place in renal or vascular disease. Page[3] injected 400 cc. of blood from women with eclampsia and severe pre-eclampsia into normal pregnant women, who showed no rise in blood pressure or unusual symptoms. Dieckmann[1] confirmed this experiment. The ischemic theory has promising possibilities; it is compatible with overdistention of the uterus. However, other factors must be involved.

The signs and symptoms of pre-eclamptic toxemia are an increase of the blood pressure (especially diastolic) over pre-existing levels and the appearance of albuminuria in the last trimester of pregnancy. In the great majority of cases, these findings are preceded by a marked increase in weight from fluid retention. One still sees an occasional case of "dry eclampsia," which usually occurs in very young primiparas and is a virulent form that rapidly develops into coma and convulsions. These patients apparently do not have fluid retention or a post-partum diuresis.

Sixty to 70 per cent of all pregnant women have some form of fluid retention or visible edema, or both. The most common form is the dependent edema of the legs, largely mechanical in origin owing to increased venous pressure. It is not important in relation to the development of pre-eclamptic toxemia. Edema of the face, hands and abdominal wall is of more significance in relation to pre-eclampsia. The most serious of all types is fluid retention in the extravascular tissue space, which may or may not be accompanied by clinical edema. This can only be judged by weight gain.

Pre-eclampsia does not develop in all women who retain fluid, although such patients are most prone to pre-eclampsia. There is as yet no method of determining which women will become toxic and which will not. Therefore, it is essential to treat all such women as cases of potential toxemia. In the average patient fluid retention can be well controlled by a proper diet. If it is not controlled, it shows either that the patient is not on the proper diet or that toxemia may be developing.

The hypertension in pre-eclampsia must be judged by a different standard from that of essential hypertension and renal disease. The diastolic pressure is the more important index. A systolic pressure of 140 to 150 may indicate impending eclampsia. A rise in blood pressure to 160 systolic, with a diastolic pressure of over 100, may mean a very sick patient. Patients with renal disease and essential hypertension may have a higher blood pressure without serious trouble.

The amount of albumin in the urine is of definite prognostic significance. The finding of a +++ to ++++ test for albumin indicates advanced toxemia. In the early stages of pre-eclampsia, albumin is the only abnormal finding in the urine. In the severe stage, and in eclampsia, casts and red blood cells may appear in the sediment. There is no demonstrable change in renal function in mild pre-eclampsia.

Early pre-eclampsia causes very few and sometimes no symptoms. It may be entirely an accidental finding in the physician's office. Lethargy and mild headaches may be the first complaints. As symptoms develop with the progress of the disease, they tend to be cerebral and visual. A frontal headache is the most common. Visual disturbances such as double vision and spots before the eyes are also frequent. Occasionally, blurring or complete loss of vision is the first subjective symptom. The development of symptoms in addition to preexisting signs is always of serious import. The patient may be rapidly approaching the eclamptic state. This is particularly true of epigastric pain of the girdle type. In patients who have had no prenatal care it is frequently the development of the symptoms that first brings them to the doctor; at this point they may be acutely ill.

In early or mild pre-eclampsia, there is no important change in the blood chemical constituents. In certain areas of poor economic status and diet, patients may show a hypoproteinemia. Correction of this will improve the patient's condition. This does not seem to be a factor in most patients with toxemia. The nonprotein nitrogen is within normal limits. The blood urea nitrogen is low in pregnancy but does not change in toxemia. A rise in the blood uric acid is characteristic of some of the severer forms, but this is not always present. Hematocrit and hemoglobin estimations are usually within normal limits. With the progression to severe pre-eclampsia or eclampsia there may be marked changes in the blood chemical findings as well as signs of renal failure.

The pathology of severe pre-eclampsia and eclampsia is primarily renal, cerebral and hepatic. The typical renal lesion is a glomerulonephrosis, which is apparently secondary rather than primary. This lesion is not found in mild cases but only when the disease is severe. This has been the finding in the Mallory Institute of Pathology, where many cases have been studied. Also in that institution, typical lesions of peripheral hemorrhagic necrosis of the liver have been found in only 50 per cent of all cases of eclampsia. The cerebral pathology consists of cerebral edema, followed in many of the severe cases by macroscopic and microscopic hemorrhage. It is also believed in this institution that there is a characteristic premature aging of the placenta in cases of pre-eclamptic toxemia that is not found in the other hypertensive states. Bartholomew and Colvin[4] describe acute hemorrhagic infarcts of the placenta, which they regard as grossly characteristic of pre-eclamptic eclampsia. I am inclined to agree with their observations. There appears to be a gross differentiation between the old yellow and white infarcts of the placenta in chronic nephritis and the fresh hemorrhagic infarcts in pre-eclamptic toxemia.

The treatment for pre-eclampsia is the prevention of its occurrence. There is no specific treatment for the disease once it has developed except termination of pregnancy. During recent years, with improved prenatal care, and especially, with the realization of the importance of sodium retention, there has been a marked decline in the incidence of pre-eclampsia in most obstetric clinics. In clinics where the patients avail themselves rarely or not at all of prenatal care, pre-eclampsia is still frequently seen.

Dieckmann[1] has demonstrated the importance of sodium retention in the development of pre-eclampsia. The ingestion of large amounts of sodium will rapidly put weight on the normal pregnant woman. Elimination of sodium will reduce this weight gain. Intravenous injection of sodium into the pre-eclamptic woman will cause an increase in the edema and blood pressure, a marked increase in proteinuria and an increase in symptoms. A falling blood pressure in definite relation to the effectiveness of diuresis is a common observation in an obstetric ward. The blood pressure rises again after diuresis if the patient again begins to retain fluid and electrolytes.

The treatment of pre-eclamptic toxemia remains confused and unsatisfactory. The method must be adjusted to the individual case, depending on the severity of the disease, its rate of progression, any underlying renal or cardiac lesion and duration of the pregnancy. The fundamental and primary object of treatment is to cause an elimination of retained fluid. The amount and extent of diuresis is probably the soundest factor on which to base prognosis. The hypertension and albuminuria will respond favorably to any successful diuresis.

Laboratory studies are not of great help in the mild or early case of pre-eclamptic toxemia. The ability of the kidney to concentrate and dilute urine is the most sensitive and helpful test of renal function. Any impairment of this function suggests underlying renal damage and makes the prognosis

more serious. The blood uric acid and serum proteins should be checked. A low protein must be replaced. A rise in uric acid is an indication of pre-eclamptic toxemia. The blood should also be checked for any evidence of anemia, immediate treatment being instituted when indicated.

In discussing treatment of this condition, one must divide the cases into mild and severe. When mild there is sufficient time for treatment and a reasonable expectation of sufficient improvement so that the patient may continue with her pregnancy for a reasonable period. In the severe case, impending eclampsia may force one's hand in a matter of days or sometimes hours. It is well to remember, especially in the mild case, that the duration of pre-eclampsia has a greater influence than its severity on the development of permanent renal or vascular damage. This is perfectly reasonable when one considers that the longer arteriolar spasm is present, the more permanent the change in the vessel walls.

On admission to a hospital, the patient should be put immediately to bed and kept at complete bed rest. A mild sedative is helpful in calming her fear and restlessness. She should be kept as quiet as possible, with a minimum of visitors. A chart of fluid intake and output should be immediately established, and all precautions taken that this is accurately kept. Because of the loss of fluid through the skin and lungs, particularly during hot weather, this chart will not be too accurate, but it should show a negative fluid balance if an efficient diuresis is taking place. A low-sodium diet is instituted, 200 mg. being the lowest practical objective. This diet must be carefully controlled, and all precautions taken that the patient receives no other food. In addition to low sodium, the patient must be given the necessary proteins, minerals and other substances of a well rounded diet.

The question of fluid intake in these patients has been much debated, and there is no common agreement. After many years of restricting fluids, I have changed to giving fluids as desired. No definite clinical change has been noted on the two regimens. The patients are more comfortable on free fluids, which, theoretically, may help flush the retained electrolytes through the kidneys.

Diuretics are a helpful adjunct to treatment. Ammonium chloride is the most commonly used and is both efficient and safe. Mercurial diuretics are also used and appear to be somewhat more efficient. The danger of mercurial diuretics has been overstressed. However, if a patient has definite renal failure, ammonium chloride may be safer.

Under this regimen at the Boston City Hospital, a weight loss of 4 to 6 pounds in three or four days is considered to indicate a good prognosis. Frequently, this weight loss is exceeded in this period. In a week a patient should lose 10 to 15 pounds. The prognosis is based on this daily weight loss, which correlates closely with the regression of the other signs and symptoms. As mentioned above, the actual loss is a more accurate gauge than a chart of fluid intake and output.

A patient who responds promptly to treatment within five days to a week is considered safe to discharge home under careful supervision. She is given a printed list of a proper low-sodium diet. The dangers and warning signs of her condition are explained to her. She is seen at home by the visiting nurse and every few days at the clinic.

Patients with early or mild toxemia generally respond well to treatment; those who do not are reclassified in the severe group and treated accordingly. In the treatment of pre-eclamptic toxemia, one must learn to differentiate the mild and the severe case. Sometimes, this is self evident, and at other times the response to treatment is the deciding factor. The blood pressure can be quite misleading. Patients have gone into convulsions with a systolic pressure of 140 and lower. One must always remember that a mild hypertension may be of serious import in toxemia although not dangerous in the other hypertensive syndromes. A pre-eclamptic toxemia with a blood pressure of over 160 systolic and particularly over 100 diastolic must be considered a serious case. The measurement of the albumin in the urine is important, since large amounts indicate severe toxemia. In addition, the symptoms are extremely significant in the estimation of a given case, any marked cerebral symptoms (severe and persistent headache, mental lethargy and confusion) or blurring or loss of vision being extremely serious. Epigastric pain, particularly of the girdle type, frequently warns of impending eclampsia.

Marked weight gain, with a rapid appearance of signs and symptoms, indicates an acute and fulminating toxemia. In certain primiparas under twenty a severe toxemia may develop, progressing to eclampsia in a few days or a week.

Once a case has been classed as severe pre-eclampsia, the termination of pregnancy may have to be considered. It should be considered immediately if the baby is sufficiently mature to survive.

The method of delivering the patient must depend on the individual case. Time is an important factor. A long, inefficient labor or an unsuccessful induction may precipitate eclampsia. Induction of labor should not be attempted unless the presenting part is well engaged and the cervix most favorable. Since severe toxemia develops in most primiparas and many multiparas before the cervix is favorable for induction, cesarean section is indicated in such cases.

The question whether a patient with severe pre-eclampsia should receive an attempt at medical treatment before the pregnancy is terminated again depends on many factors. There is definite dan-

ger in waiting, and yet in certain cases a patient's condition may be improved by a few days of medical treatment. If disease is of short duration, with rapid progression, immediate termination is probably indicated; if it has progressed slowly and is not too severe a course of medical treatment may be sufficient. It must always be remembered that post-partum eclampsia may develop if the disease has become too acute before delivery.

In addition to bed rest, low-sodium diet, diuretics and sedation, magnesium sulfate has a long and well recognized place in treatment. Primarily effective in lowering hypertension, it may be given intramuscularly in doses of 5 to 10 cc. of a 50 per cent solution. After the initial dose it may be repeated every six hours. This is helpful for temporary use but should not be given for more than a few days.

A great deal of work has been done with *Veratrum viride* and its derivatives, which are effective hypotensive drugs. A reduction of blood pressure can be obtained and maintained over a considerable period. Certain dangers in the use of these drugs must be fully understood, although the active fractions are safer than the crude drug. In a proper environment where the patients can be carefully watched and checked these drugs may be of value in trained hands. Many other hypotensive agents are appearing and are being investigated. However, it must be understood that the lowering of hypertension does not cure pre-eclamptic toxemia. In fact the question of increased anoxemia must be considered, since the hypertension may be a protective mechanism. The whole problem of hypotensive drugs offers much hope but is still an investigative rather than an accepted procedure.

Eclampsia

Eclampsia, or the development of convulsions or coma, or both, is the terminal stage of pre-eclamptic toxemia. The only successful treatment of the disease, again, is its prevention — in other words the pregnancy in patients with severe pre-eclampsia should be terminated before eclampsia develops. The recognition of this fact has caused a marked diminution in the incidence of eclampsia throughout the United States. Also, public realization of the importance of prenatal care has brought most patients to the doctor in time for the preceding pre-eclampsia to be recognized and treated.

In the great majority of cases in which a patient is under observation the physician has sufficient time to recognize and treat an impending toxemia, terminating the pregnancy if necessary, before eclampsia develops. There is still the occasional case of acute fulminating toxemia in which toxemia may develop, convulsions appearing in a few days. It is this type of eclampsia that is still seen. In addition, some patients with no prenatal care still arrive in the hospital in convulsions. The doctor who has a patient under observation for pre-eclampsia and allows eclampsia to develop by delaying the termination of pregnancy is permitting the patient to progress from a condition with a mortality of less than 0.5 per cent into one in which the mortality is from 15 to 25 per cent.

Impending signs of developing eclampsia in the pre-eclamptic patient were mentioned above. Any great rise in blood pressure or increase in albuminuria, headache, visual symptoms, mental confusion, or epigastric pain, especially of a girdle type, constitutes a sign of impending eclampsia. In addition, any patient who does not show definite improvement under hospital treatment is a potential candidate for eclampsia.

In severe pre-eclamptic toxemia and especially in eclampsia, definite signs of renal failure appear. Casts, blood, and large amounts of albumin are present. Anuria and oliguria frequently develop. A rise in nonprotein nitrogen and other changes in blood chemical constituents accompany renal failure. The systolic and diastolic blood pressures rise to high levels. The appearance of convulsions and coma completes the picture. The patient is now critically ill. The more rapid the convulsions and the longer they continue, the more serious the prognosis. Deep coma between convulsions, anuria and pulmonary edema are all of critical import. A rise in temperature, pulse and respirations is an ominous sign.

The immediate treatment of a patient with eclampsia is the control of the convulsions and coma. No immediate attempt at delivery should be made. However, once the convulsions are under control, immediate delivery may be necessary to save the patient's life.

On admission, the patient should be placed in a quiet, cool, dark room. A nurse or doctor should be in constant attendance. Nothing should be given by mouth, and a suction apparatus must be available. The patient should be disturbed as little as possible by examinations or other procedures, 15 mg. (¼ gr.) of morphine should be given immediately. Magnesium sulfate, 5 to 10 cc. of 50 per cent solution, is given intramuscularly. This can be followed in an hour by barbiturates or chloral hydrate. The sedation should be repeated at sufficiently frequent intervals to quiet all restlessness. Administration of magnesium sulfate may be repeated every six hours. Chloroform is still useful in controlling the actual convulsions. Trilene may prove to be a safer substitute. If oliguria is severe or anuria develops, hypertonic dextrose in distilled water may be given intravenously in 5 to 20 per cent solution.

Once the patient has recovered from the convulsions and become relatively conscious, the question of delivery should be considered. In a multipara with a favorable and ripe cervix, rupture of the membranes may be indicated. If this procedure is performed, the medical treatment

must be continued as before. The patient should be allowed to go into labor, with normal or low forceps delivery. If the cervix is unfavorable, and in all primiparas, cesarean section is indicated. After any type of delivery, the medical treatment should be continued over a period of days until the patient is definitely improved.

As shown by Dieckmann[1] the interval between the first convulsion and delivery is extremely important. The longer this interval, the higher the maternal mortality. On the other hand, attempted delivery during the convulsive stage carries the highest mortality. Dieckmann[1] recommends delivery within six to eight hours if possible. However, this interval must be prolonged in certain cases. It is a matter of the best obstetric judgment to decide the safest moment to rupture the membranes or perform a cesarean section as indicated.

In post-partum convulsions, the cause of the disease has already been removed. This is less serious than ante-partum or intra-partum convulsions. The same treatment should be given although the termination of pregnancy has already been accomplished.

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MEDICAL INTELLIGENCE

SPONTANEOUS HEMORRHAGE WITHIN THE RECTUS SHEATH*

Report of a Case

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THE occurrence of hematomas within the rectus sheath, although relatively rare, has been sporadically described in the medical literature since the time of Hippocrates and Galen. Most of the reports[1-4] emphasize the difficulty of accurate preoperative diagnosis and state that this condition is rarely thought of in the differential diagnosis of painful abdominal tumors.

Most of the reported cases have occurred as a result of direct trauma.[1] Critical evaluation of those that were thought to be spontaneous revealed a coincidental occurrence of sudden stress or strain on the abdominal wall. These included coughing, sneezing, sudden turning in bed and coitus. A weakened abdominal wall during pregnancy has also been found to be a cause.[5, 6]

In the case reported below, no external trauma or strain was known to have occurred. The patient, however, suffered from severe hypertension, arteriosclerosis, myocardial damage and complete heart block. It is a distinct possibility that the hemorrhage was due to altered circulatory dynamics in a vessel weakened by atherosclerosis. Cullen and Brödel[2, 3] have also reported a case in which heart disease was assumed to have been an etiologic factor.

Case Report

A 65-year-old woman was admitted to this hospital on April 21, 1953, complaining of dyspnea and edema as well as intermittent pain in the upper abdomen for 15 years. The pain was associated with epigastric distress, but there was no jaundice, reversed peristalsis or relation to food. Recently, a gallbladder series taken by a private physician was reported as negative.

The past history included arteriosclerotic and hypertensive heart disease for at least 15 years, with chronic congestive heart failure necessitating a daily ration of digitalis. Two months before admission the patient became bed ridden because of dyspnea and progressive edema of the lower extremities.

Physical examination showed an obese woman in no acute distress. Examination of the eyes, ears, nose and throat revealed no abnormal findings. The neck veins were somewhat congested, with slow venous pulsations. There was no adenopathy, and the thyroid gland was not enlarged. The thorax was symmetrical. The breasts were pendulous. Except for a few moist inspiratory rales at both bases, the lungs were clear to auscultation and percussion. The heart was enlarged, the border of dullness extending downward and to the left. A definite systolic murmur was heard at the apex. The abdomen was obese, and a well healed scar from a previous hysterectomy was noted. There was no tenderness or rigidity, and no masses or organs were palpable (this was confirmed by several examiners). The lower extremities were moderately edematous.

The blood pressure was 230/120; the pulse was 40 and irregular.

An electrocardiogram demonstrated coarse fibrillation, with complete auriculoventricular block, at a rate of 47. A right-bundle-branch block was also present.

Early in the morning after admission an extremely severe tearing pain in the right lower quadrant suddenly developed. There was no associated nausea or vomiting. Fever was of low grade, the temperature running around 100°F. The blood pressure was 230/140; the pulse rate remained at 40 and was irregular. Examination of the abdomen revealed a tender, nonmovable, hard mass about 11 cm. in diameter in the right lower quadrant. The abdominal wall was not rigid.

Urinalysis revealed a ++ test for albumin and a negative test for sugar, 1 to 3 white blood cells per high-power field, occasional red blood cells and rare epithelial cells in the sediment. Ex-

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amination of the blood disclosed a red-cell count of 3,220,000, with a hemoglobin of 12.5 gm. per 100 cc., and a white-cell count of 5600, with 66 per cent neutrophils, 3 per cent stab forms, 28 per cent lymphocytes, 1 per cent eosinophils and 2 per cent monocytes. A plain film of the abdomen was normal.

The mass grew in size, and within a few hours it reached the dimensions of 13 by 15 cm. No pulsation was felt at any time. Many opinions of the character of this mass were expressed by the medical and surgical staff. The differential diagnosis included volvulus, intussusception, cancer of the cecum with perforation, twisted ovarian cyst and appendiceal abscess. Surgical intervention was deemed imperative.

At operation a low right-rectus-muscle incision was made. The subcutaneous tissues appeared edematous. When the outer rectus sheath was incised a hematoma the size of a grapefruit, containing intermingled muscle and necrotic tissue, was found. The blood clot and necrotic mass were evacuated, and all the oozing points ligated. Despite careful search the inferior epigastric vessels could not be identified. The underlying peritoneum and viscera were normal to inspection and palpation. The wound was packed with Gelfoam, and a drain placed within the rectus sheath. Chromic sutures were used for closure. A few hours after the operation, the dressings were found to be saturated with blood. The source of the bleeding could not be identified. A compress binder was applied. The patient died shortly afterward. Surgical intervention was deemed imperative in spite of the poor general condition of the patient.

Discussion

The preoperative diagnosis of hemorrhage within the rectus sheath can easily be made when a history of violent or direct trauma is elicited.[1] There is confusion, however, when the trauma or strain is trivial. In these cases ovarian cyst, appendiceal abscess, rupture of intestinal neoplasm and other acute abdominal conditions are often diagnosed. Cullen and Brödel[2, 3] state that if the hematoma is below the umbilicus the absence of the posterior rectus sheath may permit the blood to accumulate between the muscle and the peritoneum. As a result, peritoneal irritation may occur, making the diagnosis even more difficult.

The usual symptoms consist of the sudden onset of pain in the abdomen, often associated with nausea, constipation, distention and muscular rebound elicited over a suddenly developing and expanding tumor. The rapid change in size particularly should suggest hemorrhage. The borders of the mass are always limited by the rectus sheath no matter what the size of the tumor.

The following maneuver may prove helpful in differentiating a painful intra-abdominal tumor from one within the abdominal wall. The examiner's hand should be firmly placed over the involved area with the patient supine. The patient is then asked to sit up against the examining hand. If the lesion is on the abdominal wall the mass will bulge, and the increased local pressure may produce greater pain. An intra-abdominal mass, on the other hand, will be splinted by the abdominal wall and become less marked or disappear entirely; the pain may also be somewhat ameliorated by pressure. Unfortunately, this maneuver was not attempted in the case reported above, in which no history of trauma or strain could be obtained. The associated hypertension, high pulse pressure and irregular, slow pulse may have so altered the hemodynamics of a major atherosclerotic vessel as to produce a spontaneous rupture.

Summary

The medical literature is reviewed, emphasizing the relative rarity and difficulty of diagnosing spontaneous hemorrhage within the rectus sheath.

A case is reported in which the only cause may have been altered hemodynamics. It is suggested that peripheral arteriosclerosis be added to the possible etiology of this condition.

Attention is called to the necessity of including spontaneous hematomas of the abdominal wall in the differential diagnosis of acute abdominal conditions, and aids to physical diagnosis that may prove helpful are suggested.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*

VIRGINIA W. TOWNE, *Assistant Editor*

CASE 39531

PRESENTATION OF CASE

A sixty-nine-year-old former post-office employee entered the hospital complaining of loss of weight and progressive weakness.

The patient had felt well until eighteen months previously, when he noted the gradual onset of weakness of his legs that progressed to the point where, six months before admission, he found it necessary to retire. About three months later the weakness was accompanied by pain in both calves and thighs and the lower back. Relief was obtained only by lying or sitting. About nine months before admission he began to experience some urgency of urination and some burning but no difficulty in starting his stream. He also began to have nocturia (two times). About four months before admission he noted intermittent ankle swelling at intervals of about two weeks. A month later he was examined in another hospital because of "colds in the chest." Physical examination was negative except for dullness and rales at the left-lung base and a blood pressure of 200 systolic, 120 diastolic. The urine had a specific gravity of 1.022 and was otherwise normal. Routine blood studies were normal. A roentgenogram of the chest gave findings in the left lower lobe consistent with bronchiectasis. On a subsequent visit about two weeks later the blood pressure was recorded as 180 systolic, 90 diastolic. Two months before admission the patient began to expectorate yellow or greenish mucoid material, especially at night. He denied orthopnea or dyspnea on effort. About a month later marked anorexia developed, and his weight fell from a normal of 130 to 109 pounds. Despite generalized weakness he had not experienced malaise, nausea, vomiting, diarrhea, fever, chills or night sweats.

He smoked moderately and had been in good health except for a slight cough, "bronchitis" for many years and "arthritis" of both hands.

Physical examination showed a man who appeared chronically ill but was in no acute distress. The neck veins were not distended. The lungs were clear except for some loud, bubbly rales in the left-lung base. The heart was of normal size, with a Grade 2, early systolic murmur. The abdomen was soft but slightly tender over the right epigastrium. No organs or masses were felt. The prostate was two or three times the normal size and firm and symmetrical. Extension of the metacarpophalangeal joints was limited.

The temperature was 100°F., the pulse 100, and the respirations 24. The blood pressure was 150 systolic, 90 diastolic.

The urine had a specific gravity of 1.009 and was negative for albumin, bile and sugar. The sediment contained numerous granular and hyaline casts, 5 to 10 white cells and 3 to 5 red cells per high-power field. Bence–Jones protein was absent. Examination of the blood disclosed a hemoglobin of 7.5 gm. per 100 cc., a hematocrit of 26 per cent and a white-cell count of 9000, with 86 per cent neutrophils. The fasting blood sugar was 108 mg., the nonprotein nitrogen 72 mg., and the total protein 5.5 gm. per 100 cc., with an albumin-globulin ratio of 1.0; the sodium was 132 milliequiv., the chloride 95 milliequiv., the potassium 7.0 milliequiv., and the carbon dioxide 25 milliequiv. per liter. The stool guaiac test was negative. Sputum cultures grew out beta-hemolytic streptococci on several occasions and also *Escherichia coli*. Blood culture (two flasks) was negative. An electrocardiogram revealed a sinus rhythm with a rate of 70 and some auricular premature systoles, a PR interval of 0.16 second and a QRS interval of 0.08 second. The P waves were normal, the R waves were fair sized, the T waves and ST segments normal in Lead 1 and the T waves peaked in Leads 2, 3, aVF, V_3, V_4, V_5 and V_6. X-ray films of the bones were negative for metastases. A plain film of the chest demonstrated cystic areas in the left lower lobe and lingula consistent with bronchiectasis (Fig. 1). A gastrointestinal series disclosed a rounded, smooth filling defect measuring 1.5 cm. in diameter at the midportion of the lesser curvature. No other intrinsic disease was seen in the duodenal cap or small bowel. A second gastrointestinal series confirmed the defect seen previously in the stomach. Barium-enema examination was negative.

From the time of admission the patient went gradually and steadily downhill. A bone-marrow biopsy revealed lack of hyperplasia of the erythroid series (myelocyte-erythrocyte ratio of 5:1), with abundance of stored iron. A blood smear showed a normocytic, normochromic anemia but was otherwise not remarkable. A bronchoscopic examination was negative except for the finding of purulent sputum coming from the orifices of the basilar segments of the left lower lobe. Shortly after admission the patient was started on penicillin, strepto-

mycin and sulfadiazine. Later on he received potassium iodide, which was discontinued because of vomiting and a vesiculopustular dermatitis. Urine cultures grew out nonhemolytic streptococci and later on *Pseudomonas aeruginosa* (*Bacillus pyocyaneus*) and abundant *Proteus vulgaris* (*B. proteus*). A cystoscopic examination failed to give evidence of obstruction to urinary flow, and a retrograde pyelogram was normal, with good filling of the calyceal

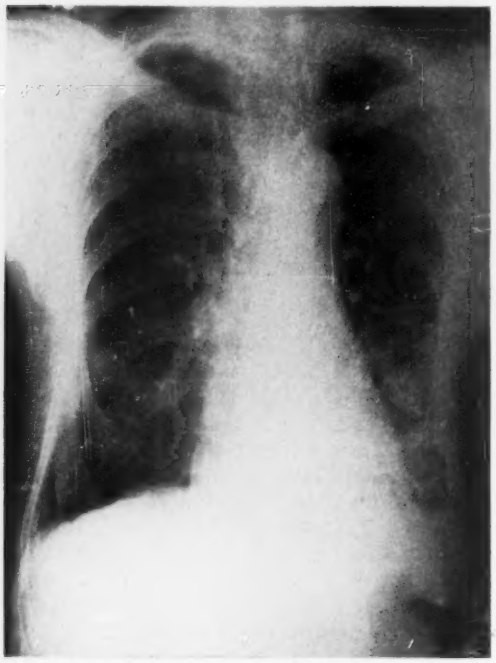

FIGURE 1. *Roentgenogram of the Chest, Showing Extensive Cystic Bronchiectasis of the Left Lower Lobe and Lingula.*

system and renal pelves. Separate urine specimens taken from the ureters grew out a few *Esch. coli* on both sides. In nine subsequent urine examinations, the specific gravity was above 1.010 only once, when it was 1.020; two specimens gave a ++ test for albumin, but the others were negative. The sediment was variable, on one occasion being considered negative; more often, however, it contained occasional to 2 to 10 white cells per high-power field. The greatest number of white cells, 30 to 50, were found one day after cystoscopy, when there were a similar number of red cells. Casts and red cells were otherwise not noted in routine urinalyses, after the admission urine specimen. However, on the twenty-seventh hospital day (two days after cystoscopy) a consultant who examined the urine found a trace of albumin, and 3 to 6 red cells, 10 to 30 white cells, many coarsely granular casts and a rare hyaline cast per high-power field. The stool guaiac test was negative on four occasions and positive on another. The patient was given several transfusions, and the hemoglobin ranged from 5.5 to 8.5 gm. per 100 cc., and the hematocrit from 25 to 34 per cent. The white-cell count rose by the twenty-fourth hospital day to 21,000, with 78 per cent neutrophils, but later fell to 10,000 and was 13,000 two days before death. On the fifteenth, twentieth, twenty-fourth, twenty-seventh, thirty-seventh and forty-first hospital days the serum nonprotein nitrogen was 104, 120, 50, 94, 104, and 160 mg. per 100 cc. respectively. The alkaline phosphatase was 6.6 units on the twelfth and 16.7 units per 100 cc. on the thirty-seventh hospital day. The sodium and chloride did not vary markedly from the normal; however, the carbon dioxide reached 18 milliequiv. and the potassium 7.0 milliequiv. per liter. Other studies included a Congo-red test, which showed 65 per cent retention in the serum, a serum lipase of 0.4 cc., a serum iron of 110 gamma per 100 cc. and a urinary urobilinogen of 0.5 Ehrlich units. A tuberculin skin test was negative in a dilution of 1:100,000; a lumbar puncture was also negative.

The temperature curve was irregular throughout most of the hospital stay, with frequent elevations to 101 or 102°F. Although during the first month the blood pressure was about the same as that on admission, it subsequently tended to be higher, with an average of about 180 systolic, 100 diastolic, and on the thirty-ninth hospital day was recorded by the nurse as 185 systolic, 90 diastolic. The urine output averaged about 750 cc. a day. Terminally, the appetite became poor, and vomiting increasingly frequent until finally nothing taken by mouth could be retained. The patient had diarrhea and became lethargic and confused. He died quietly on the forty-fourth hospital day.

Differential Diagnosis

DR. MARIAN W. ROPES*: One obvious fact about this case is that many systems were involved — the renal, the skeletal, probably the nervous, the vascular, the pulmonary, the gastrointestinal and the hemopoietic, with an anemia. The skin was also involved, but I shall leave that out because I think the dermatitis was associated with the iodine that was administered. One thing certain is that the patient had renal failure, from which he died. What the underlying cause of the renal failure was and how the diseases of the many systems were interrelated are much less certain. It seems probable, from the course and intertwining of the development of the various conditions, that the involvement of many if not all of the systems was related.

Among the various groups of conditions one has to consider in such cases are generalized infections.

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There is no evidence of an acute generalized infection. There were, to be sure, *Esch. coli* in the sputum, which is peculiar unless antibiotics had been given. Although there was no reference to antibiotics, I assume that the patient received them at some time and that the *Esch. coli* in the sputum was secondary to them. In any event I cannot connect it with the generalized disease. One infectious condition that has to be mentioned whenever there is a great deal of renal and pulmonary involvement is tuberculosis; however, although both the renal and pulmonary disease were typical of tuberculosis the remainder of the picture was not. Moreover in a patient as sick as he was, the old tuberculin test even in a 1:100,000 dilution should have been positive.

The second group to consider is cancer. I think a primary gastrointestinal cancer or a pulmonary or prostatic cancer with generalized involvement is very unlikely. I see no evidence for it. A condition like multiple myeloma, however, always has to be considered when severe renal disease and skeletal symptoms are present together. The renal disease, with its relative lack of urinary abnormalities, is consistent with either the renal involvement of myeloma or the amyloidosis secondary to myeloma. The rest of the conditions would not be well explained by it. It is conceivable that he had myeloma and in addition had a superimposed pulmonary disease but it is unlikely. The very low globulin would be extremely unusual, but without any fractionation it does not rule out myeloma. The alkaline phosphatase interests me; I cannot interpret it but I wonder if it puts any more weight on bone disease than I had considered, or on liver disease.

Dr. Bernard M. Jacobson: Won't you say a kind word for the bone marrow?

Dr. Ropes: I am glad you mentioned it. Of course, that is another point against multiple myeloma. I am going to rule multiple myeloma out, but I was trying to include all the factors that might give some weight to it. I can think of no other generalized cancer that I should consider very seriously.

Another generalized disease one must consider is amyloidosis. Ordinarily, when the renal disease is so severe, it is the so-called secondary amyloidosis (if it is at all legitimate to separate the types). Amyloidosis may be secondary to myeloma or tuberculosis, but I have already said that I do not think he had either of those diseases. The secondary form might theoretically have given him the symptoms and signs of renal disease and possibly the gastrointestinal lesion, although I have never heard of an actual ulcer associated with amyloidosis. I think it is very unlikely that this was the disease.

Among the so-called generalized connective-tissue diseases, another group of generalized diseases that must be included, the one I should consider most seriously is polyarteritis nodosa. I always hesitate even to bring up this diagnosis because I think it is brought up far too often, is usually proved not to be the diagnosis and is used too often as an easy term to put on diseases that we cannot explain. Yet many features in this patient are entirely in accord with it. Severe renal disease and hypertension occur in at least 80 per cent of the cases according to most observers. Renal changes with polyarteritis are so varied that it is impossible to say that those in this patient were not consistent with it; to be sure, there were slightly fewer changes in the urine than I should expect, but I am certain that that does not rule out polyarteritis. It is obvious that he had some superimposed renal infection, but I doubt if the whole renal disease was secondary to renal infection. Death from renal failure is common in polyarteritis nodosa. It is reported to be much more frequent in men than in women. Skeletal or nervous involvement or both are found in the disease. I am given very little information about the joint disease. This patient had metacarpophalangeal joints that could not be extended. Chronic low-grade joint disease in polyarteritis is not uncommon. The leg symptoms are hard for me to interpret; they could have been on a vascular, on a neuritic or on a skeletal inflammatory basis. I see no way of differentiating. The great relief from lying down and lack of use puts more weight on the vascular basis; however, the degree of weakness perhaps gives more weight to the neurologic and the pain extending up to the back to the skeletal basis. Perhaps this would be a good time to look at the x-ray films.

Dr. Joseph Hanelin: The principal radiographic findings are in the chest. There is evidence of a severe cystic bronchiectatic process in the lower portion of the left lung chiefly confined to the lower lobe, which is reduced in size (Fig. 1); there is perhaps some bronchiectasis on the right, but this is not very marked and might even be questioned from the available films. An area of density at the apex is probably due to old scarring. The lungs are emphysematous. The heart does not seem particularly enlarged. Approximately six weeks later, six days before death, the heart appears to have increased in size. There is evidence of more retained secretion in the left lower lobe and what may be an area of atelectasis in the medial portion of the right lower lobe. The lesion of the stomach is a very small defect near the lesser curvature above the angle that is probably clinically unimportant; it looks as though it may be a small adenomatous polyp or a leiomyoma. The skeleton is moderately osteoporotic. Of some help perhaps in explaining the leg pain is the extensive calcification of the branches of the femoral arteries in the upper thighs. The arthritis is mild and chiefly hypertrophic. There is no change in the sacroiliac joints that I should interpret as rheumatoid arthritis.

Dr. Ropes: I am afraid the x-ray films do not help. The pulmonary changes are much more

cystic than I had visualized from the record; therefore, it is less likely that they represented a manifestation of polyarteritis nodosa. Pulmonary symptoms such as asthma and bronchitis are common in polyarteritis nodosa. Bronchitis is never specific; usually, the changes are microscopic unless there is also severe asthma. There have occasionally been reports of small areas of consolidation with actual abscess formation and varying amounts of bronchiectatic changes in patients with polyarteritis nodosa in whom the vessel changes were found in the lung. This is not common and is not characteristic of the disease. The stomach changes interest me, in terms of the possible diagnosis of polyarteritis nodosa. I do not know how often ulceration in the stomach has ever been recorded in cases of polyarteritis nodosa. Ordinarily, the ulcerations and the hemorrhagic changes occur in the intestine, but in one series reported from the Mayo Clinic by Wold and Baggenstoss* there were actual small gastric ulcers in the base of which were small vessels with periarterial changes. Whether these were due to the polyarteritis or not I do not know.

To sum this case up I should say that the one definite fact was that the patient had severe renal disease and that he died of renal failure. Of the diseases that might involve all these systems I think that polyarteritis nodosa has more chance of explaining the findings. It seems probable that the pulmonary disease was separate, with slowly developing bronchiectasis and cystic changes.

Dr. Arthur S. Pier, Jr.: Could pyelonephritis alone have caused this?

Dr. Ropes: The renal disease could surely have been pyelonephritis. I think he had some renal infection but I tried to relate the diseases in the various systems. It is possible that he had separate involvement of each one of the systems, with at least three or four diseases.

Dr. Jacobson: Do you think the severe anemia could be explained by the renal insufficiency and sepsis?

Dr. Ropes: I think the combination of the severe systemic disease and the severe renal disease would explain the anemia.

Dr. Benjamin Castleman: For a long time the physicians on the ward considered some form of carcinoma, first of the lung and then of the stomach; after he was in the hospital for a longer time, they got away from that diagnosis. I believe one physician mentioned polyarteritis nodosa.

Clinical Diagnoses

Probable metastatic carcinoma.
Uremia.
? Polyarteritis nodosa.
Bronchiectasis.

*Wold, L. E., and Baggenstoss, A. H. Gastro-intestinal lesions of periarteritis nodosa. *Proc. Staff Mtg., Mayo Clin.* **24**:28-53, 1949.

Dr. Marian W. Ropes' Diagnoses

Polyarteritis nodosa, generalized, with renal involvement.
Uremia.
Bronchiectasis.

Anatomical Diagnoses

Polyarteritis nodosa, generalized, with marked renal involvement.
Pericarditis, acute, uremic.
Bronchiectasis.

Pathological Discussion

Dr. Castleman: Autopsy showed that the patient died of renal failure, with a characteristic uremic pericarditis. The lung lesion, as Dr. Ropes predicted, was severe bronchiectasis, involving the left lower lobe and lingula of the upper lobe. Microscopical sections of the kidneys demonstrated many thrombosed arteries in which the elastic tissue was destroyed, and others with so much inflammatory reaction with destruction of the entire wall that aneurysmal out-pocketings were formed — findings very characteristic of polyarteritis nodosa. The kidney parenchyma, as a result of numerous arterial changes, disclosed severe inflammatory reaction throughout — more of a pyelonephritis, although many of the glomeruli were necrotic as well. The arterial changes were not limited to the kidneys, similar findings being found in almost every organ of the body, including the lungs. In the submucosa of the stomach was a vessel with a similar appearance, but I do not believe this was the lesion that was observed on x-ray examination. The arteries in the muscles also had some changes, which may account for some of the weakness of the legs. The prostate showed hypertrophy with some inflammation. The contractions in the hands were Dupreytren's contractures.

Dr. Jacobson: What was the color of the bone marrow?

Dr. Castleman: The bone marrow was quite red and showed hyperplasia of the red-cell elements.

Dr. Hanelin: Was the lesion in the stomach identified at autopsy?

Dr. Castleman: No.

Dr. Beverly W. Cobbs, Jr.: The question of polyarteritis nodosa did not occur to us until we had given this man iodine the second time (in the form of Priodax) and had seen auricular fibrillation and a precordial rub suddenly develop. We then tardily suspected that he had no ordinary iodine sensitivity. Certainly, in retrospect it seemed to us also that he had quickly become much worse after the first exposure to iodides.

Dr. Castleman: I doubt if the iodism had anything to do with the production of the arteritis,

although the fact that he was hypersensitive may have been a factor in the production of the disease.

DR. ROPES: I should agree that he must have had the arterial changes before he received the iodide, but I also think that such a patient tends to be more sensitive to various drugs.

CASE 39532

PRESENTATION OF CASE

A fifty-five-year-old Negro housemaid was admitted to the hospital because of anorexia, weight loss, weakness and dyspnea.

Ten months previously she first noticed a painless swelling in the region of the submandibular and submaxillary glands. This was associated with a "cold," weakness and a slight weight loss. A roentgenogram of this area gave no evidence of calculi. Nine months before admission she began to cough up a whitish sputum and noted severe dyspnea, without other symptoms, on climbing one or two flights of stairs. Gradually, the cough, exertional dyspnea and weakness increased, and she became anorexic. Eight months later, after several drenching night sweats and a shaking chill, she was admitted to another hospital with the diagnosis of "virus pneumonia" on the right side. She was treated with penicillin, and the temperature fell from 103°F. to normal; she was discharged after ten days although she was told that she should again be hospitalized because of "abnormal findings in the lung" on x-ray examination. Two days before entry she first noted a knifelike pain in the right posterior portion of the chest, made worse by coughing or inspiration. During this ten-month period the masses in the neck had remained about the same size. Her weight had gradually dropped from 145 to 123 pounds. There was no history of edema, hemoptysis, orthopnea or paroxysmal nocturnal dyspnea.

In the past she had always been in excellent health. There was no known exposure to tuberculosis although a year before entry she had worked for a family in which there were 3 cases of "virus pneumonia." She had been born in the British West Indies, coming to the United States at the age of eighteen.

Physical examination showed a chronically ill, tired, elderly-appearing woman with a frequent, sharp nonproductive cough. In the neck there were masses, 3 by 4 cm. and 1 by 2 cm., in the right and left submaxillary areas respectively. In addition lymph nodes varying in size from "a pea to a marble" were palpated in the submandibular, anterior and posterior cervical axillary supraclavicular, inguinal and left epitrochlear regions. In all areas the lymph nodes were firm, nontender, easily movable and not matted together. The skin was clear. The scleras were muddy, and the fundi, although poorly visualized, seemed normal. The neck was supple, and the trachea was in the midline; the thyroid gland not enlarged. The tonsils were moderately hypertrophied. Over the right midlung field posteriorly tactile fremitus was increased, and bronchial breath sounds were present in the same area and at the left-lung base. The heart was not enlarged, the rhythm was regular, and no murmurs were heard. The liver edge was smooth and was palpated 5 fingerbreadths below the right costal margin. The spleen was not felt. Pelvic and rectal examinations revealed no abnormalities, and neurologic examination was negative. Peripheral pulses were normal, and there was no clubbing of the fingers or toes.

The temperature was 99.5°F., the pulse 80, and the respirations 20. The blood pressure was 115 systolic, 80 diastolic.

The urine had a specific gravity of 1.004 and gave a ++ test for albumin. The white-cell count was 4600, with a normal differential. The hemoglobin was 9.5 gm. per 100 cc. The nonprotein nitrogen was 19 mg., the fasting blood sugar 79 mg., the total protein 8.9 gm., the albumin 3.7 gm., the globulin 5.2 gm. with an albumin-globulin ratio of 0.7, the phosphorus 3.4 mg., and the alkaline phosphatase 13.8 units per 100 cc.; the serum calcium was 9.4 milliequiv. per liter. A blood Hinton test was negative. Tuberculin skin tests were negative in the highest strength. A roentgenogram of the chest disclosed bilateral enlargement of parabronchial, paratracheal and paraesophageal lymph nodes (Fig. 1). The mediastinum, though widened, was not displaced. Peripherally the lungs were clear, but there was a small amount of fluid in the right pleural cavity. Roentgenograms of the hands demonstrated no destructive lesions of the bones. On the second hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. RITA M. KELLEY*: May I see the x-ray films?

DR. STANLEY M. WYMAN: The films show considerable cardiac enlargement, probably chiefly of the left ventricle, and a tortuous aorta, which is well seen coming down behind the heart in the overpenetrated film (Fig. 1). There is definite, somewhat lobulated fullness in the mediastinum that is quite consistent with enlarged lymph nodes as noted in the protocol. A suggestion of enlarged lymph nodes is also observed in both hilar regions. A small amount of fluid is present in each pleural space. The esophagus is not remarkable. I see no evidence of splenic enlargement, and the lower border of the liver is not included on the film.

DR. KELLEY: Do you see anything that looks like involvement of the pulmonary parenchyma?

*Assistant in medicine, Massachusetts General Hospital.

DR. WYMAN: There are some linear patchy densities, which I took to be chiefly atelectasis; however, I cannot say that they are not linear mottled infiltrations. I think atelectasis is a more logical explanation; they seem to be limited almost entirely to the lower portion of the lung, as though it was not well aerated.

FIGURE 1. *Roentgenogram of the Chest, Showing Bilateral Enlargement of the Hilar Lymph Nodes and Linear Streaking at Both Bases. The left ventricle is enlarged, and the aorta is tortuous.*

DR. KELLEY: Is there consolidation to account for the bronchial breath sounds described over the right-midlung field and the left base?

DR. WYMAN: No; I do not see any appreciable consolidation. The films of the hands are noncontributory.

DR. KELLEY: Was an electrocardiogram done on this woman?

DR. DAVID KAHN: I do not believe so.

DR. KELLEY: I am confronted with a middle-aged woman who had evidence of a chronic progressive disease involving all the peripheral, mediastinal, paratracheal and peribronchial lymph nodes, the liver and probably the pleura and accompanied by certain laboratory changes that were mostly nonspecific but were compatible with a progressive chronic disease with marked systemic changes. Although there was one episode of acute onset of chills, fever, increase in cough and sweating, I gather that these symptoms were not a characteristic feature of the long-term picture, and I am assuming that that was an isolated episode of secondary pneumonia complicating the primary underlying disease. It seems to me that the diagnosis probably lies between a chronic granulomatous process of infectious or unknown nature and a malignant condition involving primarily the reticuloendothelial system.

Among the possible chronic granulomatous diseases are the infectious ones and the nonspecific ones. I am purposely omitting chemically caused granulomatous diseases that might reproduce this clinical picture because there was absolutely no evidence that the patient was carrying on her housemaid's duties within the shadow of a beryllium-belching chimney or something of that sort. Among the infectious processes to be considered are tuberculosis and mycotic infections such as coccidioidomycosis and histoplasmosis. These diseases are described as causing generalized lymphadenopathy at times, but it would be unusual to find such symmetrical and small isolated nodular involvement of the lymph nodes without more definite evidence of intrapulmonary disease or of intermittent fever and chills. In addition there was no information that this patient had inhabited the endemic areas of either coccidioidomycosis or histoplasmosis, so I think I can discount those conditions. If this had been a tuberculous process, it would necessarily have been a protracted type of miliary blood-borne disease, which I should expect to have involved other areas in addition to the lymph nodes and liver. The absence of intrapulmonary disease is against this diagnosis. Also, the process in the lymph nodes, in that it remained isolated and discrete and had no tendency to mat together or break down during the advancing course of the disease, would be most unlike tuberculosis. Moreover, it would be unusual to find a negative or anergic type of tuberculin test in this type of tuberculosis, which is a slow one in contrast to the acute miliary type that may be characterized by a negative tuberculin test. I do not think that this was tuberculosis.

I come to one of two diagnostic choices, either a chronic, so-called granulomatous process such as sarcoidosis of unknown cause or a malignant neoplasm such as one of the malignant lymphomas. Many features of the history, physical findings and laboratory elements are against the textbook picture of either of these categories of diseases. In trying to choose between sarcoidosis and one of the lymphoma group on the basis of history we think of sarcoidosis as an indolent disease characterized by

remissions and exacerbations over a long time. The clinical complaints of the patient are usually much less severe than the pathological picture presented; we are all familiar with cases in which there are no pulmonary symptoms but the x-ray films of the chest show a great deal of involvement of both lymph nodes and pulmonary parenchyma. However, there are in the literature cases of sarcoidosis that have rapidly progressed from the first appearance of symptoms and diagnosis to death in the course of several months. There is therefore a distinct possibility that I am dealing with that kind of nonbenign sarcoidosis. Usually this type of case is characterized by marked pulmonary or cardiac involvement, either primary infiltration of the myocardium with sarcoid nodules or cor pulmonale secondary to advanced pulmonary changes with fibrosis — or both. There was no evidence for the latter type but I am somewhat heartened by Dr. Wyman's description of the enlarged heart because if that was due to sarcoid involvement, it would account for some of the dyspnea, which I cannot explain on the basis of pulmonary disease alone. An electrocardiogram might have been very helpful because the patient who has infiltration of the myocardium with sarcoidosis often shows abnormalities of rhythm with some block, occasionally complete heart block, and transient arrhythmias of various types; however, with a normal pulse that is unlikely. Without other evidence of congestive heart failure, it is improbable that all the dyspnea was on the basis of cardiac involvement alone. In this type of progressive sarcoidosis there have been cases with very pronounced dyspnea and evidence of bronchial compression by involved mediastinal lymph nodes even without infiltration of the lung. This is an unusual occurrence in sarcoidosis. On the other hand, progressive lymphomatous involvement of the mediastinum can easily present as dyspnea, increasing cough, malaise, anorexia and so forth. On the basis of history alone I think that lymphoma wins the round.

The first important physical finding is the disease of the peripheral lymph nodes. The lymph nodes were not very impressively enlarged, except the two submandibular lymph nodes, but in virtually every area they were involved by disease. They were discrete and nontender; the significant point is that their character did not change over the entire ten-month period of the progressive disease. Since I cannot visualize a lymphomatous process that would be so completely static as the disease progressed, I should choose sarcoidosis.

That the liver was enlarged, smooth and nontender is of no particular help in the choice between the two categories because the liver can be involved in either disease. The absence of a palpable spleen somewhat favors sarcoidosis. The lung signs, with bronchial breathing and so forth, which were not confirmed by the x-ray films, again, are not particularly helpful since there may be microscopic pulmonary involvement with either of these processes. Of course, there may be widespread involvement of the mediastinal lymph nodes either in sarcoidosis or in Hodgkin's disease or other lymphomas. The negative findings, including the clear skin and normal heart sounds, do not contribute one way or the other. I should be happier in assigning this to sarcoidosis if the patient had some skin lesions, but they are by no means universal. On the basis of the physical findings I am inclined more to sarcoidosis simply because of the nature of the lymph-node involvement.

In the laboratory data the low specific gravity of the urine and the albuminuria in the presence of a normal nonprotein nitrogen are not helpful since either of these processes can involve the renal parenchyma. The moderately low hemoglobin is against the usual variety of sarcoidosis. This disease, even when it involves the bone, is rarely accompanied by a profound anemia; there is usually normal or a very slight depression of hemoglobin. However, I am trying not to make a diagnosis of the common variety of sarcoidosis, and I think the progressive, severe, what might be called malignant sarcoidosis, particularly, might be accompanied by prolonged anorexia and a low hemoglobin. A hemoglobin of 9.5 gm. per 100 cc., however, is compatible with widespread lymphoma even without bone involvement. The leukopenia, which was not particularly marked, is more in line with sarcoidosis than with an advanced lymphomatous process. Was there any increase in monocytes? Patients with sarcoidosis are often described as having moderate to marked monocytosis, but that again is by no means universal.

DR. KAHN: The blood smear was normal.

DR. KELLEY: The one laboratory finding of any distinguishing character is the total protein and albumin-globulin ratio. A total protein of 8.9 gm. is a definite elevation and was due largely to the

elevation of the globulin fraction to 5.2 gm. There is no question that widespread Hodgkin or other lymphoma, particularly when it involves the liver, can cause a slight hyperglobulinemia, but it would be unusual for the globulin fraction to be that high. I think the hyperglobulinemia favors sarcoidosis, in which it is usually one of the distinguishing characteristics. On the basis of the laboratory findings I think that sarcoidosis is leading by a small margin.

The alkaline phosphatase was definitely elevated; that, too, is not particularly helpful in distinguishing the two pathologic processes. The phosphatase level suggests either intrahepatic disease or extrahepatic obstruction. Either of these diseases can involve the liver directly or cause some obstructive changes.

The next possibly helpful point is the negative tuberculin test. We are all taught that the tuberculin test is rarely positive in extensive sarcoidosis, and it has been used as one of the important differential points and one of the points in the argument concerning the etiology of sarcoidosis. On the other hand many patients with lymphoma have a negative tuberculin test; therefore, it does not help. I should not, however, want to call this sarcoidosis if the tuberculin test had been positive.

The x-ray films do not help very much. The involvement of the mediastinal lymph nodes was symmetrical, which can occur in either sarcoidosis or one of the lymphomatous processes. There was no obvious parenchymal lung involvement, which I should like to see if this was diffuse sarcoidosis causing dyspnea to this extent. The normal x-ray film of the hands is not particularly helpful since in many cases of sarcoidosis bony involvement is not demonstrable by x-ray examination; if the findings were positive, I should have another argument on the sarcoid side of the problem.

Balancing up the pros and cons, the tally is nearly equally in favor of either diagnosis. Using two main points as my specific criteria, I am in favor of sarcoidosis. These two points are the nature and changelessness of the lymph nodes over a long period and the hyperglobulinemia. I am visualizing a progressive, severe type of sarcoidosis, with dyspnea, which I must postulate on the basis of compression of the bronchi, possibly myocardial involvement.

Dr. Allen G. Brailey: The fact that this woman was a Negress is in favor of sarcoidosis.

Dr. Kahn: I think in the Army series it was seventeen times more common in Negroes than one would expect from population figures.

Dr. William H. Baker: I might mention that skin anergy to tuberculin does not differentiate sarcoidosis from lymphoma in view of the fact that it is probably as prominent in Hodgkin's disease and allied lymphomas as it is in sarcoidosis.

Clinical Diagnosis

Malignant lymphoma.

Dr. Rita M. Kelley's Diagnosis

Sarcoidosis.

Anatomical Diagnosis

Sarcoidosis.

Pathological Discussion

Dr. Kahn: The operation was a biopsy of an axillary lymph node, which was slightly enlarged. Microscopical examination confirmed Dr. Kelley's diagnosis of sarcoidosis. The lymph node was almost totally replaced by epithelioid cells arranged in a whorled fashion forming tubercle-like structures. A number of giant cells were present. An occasional whorl contained a tiny area of necrosis, but there was no frank caseation. No tubercle bacilli or fungi were found in specially stained sections; guinea-pig inoculations were negative. It is not possible from the histology to predict whether this will turn out to be a rapidly advancing case or pursue a chronic course. We have only a three-week clinical follow-up period on the patient; at that time she felt well, still had some dyspnea and had a hemoglobin of 11 gm. per 100 cc.

Dr. Wyman: I should like to re-emphasize the point that Dr. Kelley brought out: that this x-ray picture may be due to sarcoidosis, to lymphoma or even in some cases to tuberculosis.

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EDITORS GOSSIP

Four years ago the American Medical Association abandoned the annual meeting of state-society secretaries and editors in favor of a biennial gathering of editors and business managers of state journals. The second of these conferences took place in November of this year.

This was, as formerly, a two-day meeting, but with a program designed more particularly for the benefit of those physicians and business men and women who are directly concerned with the production of the 34 state, district and territorial publications that benefit from the State Journal Advertising Bureau of the American Medical Association.

As in the past the meeting took place largely under the auspices of the Bureau, which has been such a potent factor in obtaining national advertising for its brood of journals and in maintaining the high ethical standards in advertising that have been established by the councils of the Association.

Without going into details that were not at all tedious for the participants, suffice it to say that the meeting dealt with matters of pharmaceutical advertising and "therapeutic research," with printing problems, including costs and typography, with the responsibilities of medical editorship and with the management of production. As an additional attraction Paul de Kruif, Ph.D., roving editor on matters medical for the *Reader's Digest*, who has at last fallen into the good graces of the American Medical Association, talked entertainingly on his experiences and observations in writing for the public about health and certain ways of seeking it.

The *Journal*, which has been a loyal client of the Bureau and a member of its group since it was first organized, has with regret indicated its decision to pursue an independent course in the procurement of advertising. This change in policy has been the result not of any dissatisfaction with the Bureau or with the councils of the Association, for all of which it has the highest regard, but of a steady growth in circulation that has put it, in respect to its position as a medium for advertisers, in a unique category.

Despite the fact that the Committee on Publications and the editors consider it to be in the best interests of the *Journal* to terminate its business association with the Bureau as of January 1, 1954, they continue to maintain the friendliest relations with the Bureau and to consider themselves as belonging to the group in all except fiscal matters.

SCHOOL VISION TESTS

A significant forward step to end the confusion that at present surrounds the choice and application of school vision tests was taken recently when the New England Ophthalmological Society and the Section on Ophthalmology and Otolaryngology of the Massachusetts Medical Society, with the cooperation of the Harvard School of Public Health and Children's Medical Center, reported their preliminary recommendations for standards for referral of school children for eye examinations. On the

basis of a questionnaire regarding current standards used by ophthalmologists in their own offices and those advocated in the referral of children for eye examinations, as well as the choice of functions to be tested and of the personnel who should administer the tests and refer the patients, several important considerations were agreed on as a point of departure in the decision of personnel, procedure and the standards themselves.

In the first place it was recommended that a lay person — teacher, nurse or other school personnel — give the vision test; it was not considered desirable to have an ophthalmologist give the test, which is at best but a rough, incomplete examination, since a physician's report of a passing grade might give the parents a false sense of security. Secondly, every child failing the test should be re-examined (by the person who gave the first test or, preferably, by the school nurse or some other specially trained personnel) before being sent home to the parents. Thirdly, the test should be given annually. Finally, it was urged that no note be sent home advising parents of a passing grade, reports being reserved for failure to pass the test and containing that information alone.

Regarding the technic itself, the best procedure in current use was considered to be the Massachusetts Vision Test, which consists of tests for visual acuity, hypermetropia and heterophoria (investigations of stereopsis, color vision and other binocular functions were not recommended for inclusion because they would increase not only the time required but also the complexity of a procedure to be administered by laymen and might lead to an unnecessarily large number of referrals). In addition, it was advised that any child with obvious eye symptoms or reading difficulty be referred for ophthalmoscopic examination, regardless of the results of the visual test.

In passing, it was pointed out that error on the side of too many referrals is always preferable to the opposite of too few. Ophthalmologists were reminded that vision tests are not always accurate and that when a negative examination follows a referral because of a failing grade on the test, it is the duty of the doctor to explain to the parents the value of the eye examination, rather than to give the impression of needless anxiety and inconvenience.

Although the preliminary nature of the recommendations is emphasized in the report, any program to set up uniform standards for referral is welcome. Further progress will be reported from time to time, and any necessary revisions in the recommendations will be made. Meanwhile, the members of the groups participating in the study are congratulated on a noteworthy contribution to the problem of visual tests for school children. It is significant that the National Society for the Prevention of Blindness is incorporating these recommendations in its official report.

THE FRAGILE MALE

It is realized, perhaps somewhat dimly, that women in general live longer than men. The latest figures confirm this impression by indicating that the life expectancy at birth is seventy and a half years for females and sixty-four and seven-tenths years for males. Further confirmation is given by the Bureau of the Census report of July 1, 1948, indicating that there were at that time 446,000 persons eighty-five years of age and older in the United States, 255,000 of whom were females and 191,000 of whom were males.

It is evident that military action, accidents and homicides involve males more frequently than they do females, but the toll of various diseases is also higher in the male. A considerable difference in mortality between the sexes since 1900 has become progressively greater since World War I. In 1926, for example, the death rate for males was approximately 14 per cent higher than that for females, whereas in 1948 the difference had jumped to 41 per cent. Again the death rate from 1940 to 1949 declined 13 per cent for females and only 7 per cent for males. In 1949 the sex differences in mortality were quite definitely in favor of females in every age group.

It seems also that the fragility of the male appears even before birth, for in 1949 about 10,000 deaths occurred in males from congenital mal-

formations, whereas in females there were only about 8000 deaths from the same causes. In many specified diseases the same difference in mortality rate exists. Approximately 800,000 deaths from all causes in 1949 were of males and 600,000 were of females; 25,000 males and 13,000 females died of tuberculosis, 420,000 males and 320,000 females of major cardiorenal diseases, and 22,700 males and 17,300 females of pneumonia and influenza.

Numerous other causes of death — acute poliomyelitis, cancer, ulcer of the stomach and duodenum, appendicitis, accidents, suicide and so forth — show increases in male over female victims. Diabetes and diseases peculiar to females are the main conditions in which the females surpass the males, and in these great progress is being made in prevention.

In Florida, where this statistical analysis (depressing from the male point of view) was compiled,* similar figures are found. In 1949 there were 25,317 deaths, of which 14,974, or about 60 per cent, were of males. There is no intention of deprecating conditions in Florida, but one cannot help wondering about the causal factors — whether males go there to die amid the widely advertised glorious atmospheric conditions, whether dangerous conditions exist in the State, and perhaps even whether the fragile male should invade the State in such multitudes, especially in the winter months.

It seems that nature makes up for all these disasters to the male by arranging for more male babies to be born. In Florida in 1949, for instance, there were 106.2 male births per 100 female births. This rate may be neutralized to some extent, however, by the fact that in 1949 the death rate in the United States for infants under one year of age was 38.3 per 1000 for males and 29.5 per 1000 for females.

The hazards of being a male and living to an age of four score or more years are great, and perhaps the geriatricians should pay greater attention to the fragile male. An alternative would be to insist that the pediatricians carry the weaker sex for a longer period through life!

*Sowder, W. T. Fragile male. *J. Florida M. A.* 38:553, 1952.

CHILDREN'S HOSPITAL SMOOTHS THE WAY

"Johnny Goes to the Hospital" is the title of an attractive booklet conceived and distributed by the Public Relations Department of the Children's Hospital in Boston. As the brochure states, the purpose is to acquaint children and parents with the hospital and its procedures, with the object of allaying the anxiety that arises when a family is faced with possible hospitalization. Written for the child in understandable language, and attractively illustrated on each page in color, the pamphlet should do much to calm the fears and dispel the anxiety that arises when the word hospital is mentioned and the sick child is faced with the prospect of leaving home. Not only the child but also the parent can derive much comfort from the depiction of the actual experience of entering a hospital and the description of the procedure in the hospital.

The Public Relations Department deserves much credit for putting forth this booklet, as well as the Accident Handbook that was such a success in 1950. The Handbook has been translated into eight languages and has sold over 1,000,000 copies.

Dr. A. G. Walters, of Pittsburgh, has recently removed a worm, about one-eighth of an inch in length, from the right eye of a patient from Indiana.

Boston M. & S. J. *January 4, 1854*

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

DIAGNOSTIC LABORATORY SERVICES

A new edition of the booklet describing the services of the Diagnostic Laboratory has just been prepared. It is designed to inform doctors and boards of health about the tests performed by the Diagnostic Laboratory, including the newer services, and how they can be utilized effectively. It is available upon application to the Diagnostic Laboratory, 281 South Street, Jamaica Plain.

For the information of physicians and boards of health the telephone of the Diagnostic Laboratory is listed in the telephone directory under the Institute of Laboratories (JA 2-3700). The telephone number for evenings, week ends and holidays, which unfortunately is not listed in the telephone directory, is JA 2-3702. A bacteriologist is in the Laboratory on Saturdays, Sundays and holidays from 9 to 12 noon, at least. In an emergency during other hours, if no answer is obtained, State Capitol Police (CA 7-4600) should be called for telephone numbers of bacteriologists.

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR NOVEMBER, 1953

Résumé

Disease	November 1953	November 1952	Seven-Year Median
Chancroid	5	0	2
Chicken pox	616	1250	862
Diphtheria	2	0	18
Dog bite	886	624	664
Dysentery, bacillary	58	17	5
Encephalitis, infectious	1	0	2
German measles	57	62	62
Gonorrhea	174	126	206
Granuloma inguinale	0	0	0
Hepatitis, infectious	77	96	14†
Lymphogranuloma venereum	3	0	0
Malaria	1	5	5
Measles	36	83	157
Meningitis, meningococcal	9	4	4
Meningitis, Pfeiffer bacillus	5	7	4
Meningitis, pneumococcal	3	1	3
Meningitis, staphylococcal	0	0	0
Meningitis, streptococcal	0	0	0
Meningitis, undetermined	8	4	5
Mumps	931	419	461
Poliomyelitis	74	22	22
Salmonellosis	20	14	8
Scarlet fever	233*	208	290
Syphilis	178	130	183
Trichinosis	5	1	4
Tuberculosis, pulmonary	187	189	196
Tuberculosis, other forms	7	6	10
Typhoid fever	2	1	2
Undulant fever	2	2	2
Whooping cough	164	100	445

*Includes streptococcal sore throat.

†Four-year median.

Comment

Diseases below the seven-year median were chicken pox, diphtheria, measles, scarlet fever and whooping cough.

The 616 cases of chicken pox reported during November made this the lowest figure for this month since 1931.

Two cases of diphtheria reported in November show that it is no longer primarily a cold-weather disease. This is 3 cases less than the 5 reported in October, and equal to the average of 2 for the months of July, August and September.

The 36 cases of measles represented the lowest figure by a considerable margin for the month of November in the history of this state.

With one exception (November, 1952) the 164 cases of whooping cough constituted the lowest figure for the month of November in the history of this state.

Diseases above the seven-year median included bacillary dysentery, infectious hepatitis, meningococcal meningitis, mumps, poliomyelitis and salmonellosis.

The 20 cases of bacillary dysentery reported, although high for the month of November, represented a considerable drop since the peak in the early fall.

Poliomyelitis is persisting late this year, as demonstrated by the fact that the 74 cases reported was the highest number for November since 1927.

Geographical Distribution of Certain Diseases

The following diseases were reported from the communities named:

Diphtheria: Dedham, 1; Fall River, 1; total, 2.

Dysentery, bacillary: Belmont, 1; Billerica, 1; Boston, 24; Cambridge, 10; Danvers, 1; Fitchburg, 1; Ipswich, 1; Lowell, 3; Lynn, 1; Malden, 1; Melrose, 1; Springfield, 1; Tewksbury, 1; Waltham, 4; Watertown, 1; Worcester, 6; total, 58.

Encephalitis, infectious: Haverhill, 1.

Hepatitis, infectious: Athol, 1; Belmont, 3; Boston, 20; Braintree, 2; Brockton, 3; Cambridge, 4; Chelsea, 1; Chicopee, 2; Fort Devens, 2; Framingham, 2; Lowell, 1; Lynn, 5; Middleboro, 2; Newton, 1; North Adams, 2; Northampton, 1; Oxford, 1; Paxton, 2; Plymouth, 4; Quincy, 4; Richmond, 2; Shrewsbury, 2; Somerville, 1; Spencer, 1; Springfield, 1; Worcester, 7; total, 77.

Malaria: Westfield, 1.

Meningitis, meningococcal: Boston, 3; Fall River, 1; Lynn, 1; Quincy, 1; Scituate, 1; Springfield, 1; Wellesley, 1; total, 9.

Meningitis, Pfeiffer bacillus: Boston, 2; Hadley, 1; Plymouth, 1; South Hadley, 1; total, 5.

Meningitis, pneumococcal: Lee, 1; Milford, 1; Pittsfield, 1; total, 3.

Meningitis, undetermined: Chelmsford, 1; Easton, 1; Fall River, 1; Falmouth, 1; Millbury, 1; New Bedford, 2; Worcester, 1; total, 8.

Poliomyelitis: Ashby, 1; Athol, 3; Belchertown, 1; Boston, 5; Brockton, 3; Brookline, 1; Cambridge, 1; Canton, 3; Dedham, 2; Easton, 1; Falmouth, 2; Framingham, 1; Gardner, 1; Great Barrington, 1; Hadley, 1; Haverhill, 2; Holyoke, 1; Hull, 2; Leverett, 1; Lowell, 2; Lynn, 2; Marlboro, 1; Marshfield, 2; Milton, 1; New Bedford, 1; Newton, 1; North Andover, 1; Northampton, 7; North Attleboro, 1; Norton, 1; Peabody, 2; Quincy, 2; Scituate, 1; Seekonk, 1; Somerset, 1; Somerville, 2; Springfield, 2; Stoughton, 1; Waltham, 1; Warwick, 1; Wellesley, 1; Wenham, 1; Westminster, 1; West Springfield, 1; Wilbraham, 1; Winchester, 1; Worcester, 1; total, 74.

Salmonellosis: Attleboro, 1; Bellingham, 1; Boston, 3; Fitchburg, 2; Lowell, 2; Medford, 1; Quincy, 3; Revere, 1; Salem, 1; Somerville, 1; Springfield, 2; Wakefield, 1; Watertown, 1; total, 20.

Trichinosis: Boston, 2; Framingham, 1; Medford, 1; New Bedford, 1; total, 5.

Typhoid fever: Fall River, 2; total, 2.

Undulant fever: Boston, 1; Cambridge, 1; total, 2.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

New Hope for the Retarded: Enriching the lives of exceptional children. By Morris P. and Miriam Pollock. 8°, paper, 176 pp., with 29 illustrations. Boston: Porter Sargent, 1953. $4.50.

This textbook is based on a twenty years' experience with retarded children. It is intended for students and workers in the field of mental health and abnormal child psychology. The preliminary chapters, which discuss the mentally retarded child, are followed by chapters on the authors' program, their curriculum and an extensive presentation for the devices and projects used in their work. There is a special chapter entitled "Teaching 'Speechless' Children to Talk." There is also a list of psychiatric clinics for children. The monograph should be in collections on mental hygiene and mental deficiency. It is a practical book.

Cybernetics: Circular causal and feedback mechanisms in biological and social systems. Transactions of the Ninth Conference, March 20-21, 1952, New York, New York. Edited by Heinz von Foerster. Department of Electrical Engineering, University of Illinois; assisted by Margaret Mead, American Museum of Natural History, New York City, and Hans L. Teuber, Department of Psychiatry and Neurology, New York University College of Medicine. 8°, cloth, 184 pp., with illustrations. New York: Josiah Macy, Jr. Foundation, 1953. $4.00.

Ten papers on various aspects of cybernetics were read at this ninth conference. An appendix lists the references for the papers. The publishing, as usual, is excellent. The series should be in all medical libraries.

Peptic Ulcer: Pain pattern, diagnosis and medical treatment. By Lucian A. Smith, M.D., M.S., assistant professor of medicine, Mayo Foundation, and head of section, Division of Medicine, Mayo Clinic; and Andrew B. Rivers, M.A., M.D., M.S., consultant, Division of Medicine, Mayo Clinic. With a foreword by George B. Eusterman. 8°, cloth, 576 pp., with 208 illustrations. New York: Appleton-Century-Crofts, Incorporated, 1953. $12.50.

The authors, assisted by Drs. Herbert W. Schmidt, C. Allen Good and Richard R. Ferayorni, have presented a thorough discussion of peptic ulcer, with emphasis on treatment. The first chapter is devoted to history from the earliest time to the present. This is followed systematically by chapters on anatomy and pathologic anatomy, physiology, etiology, diagnosis and symptoms and treatment of the disease and its complications. References are appended to the chapters, and there is a good index. The publishing is excellent. The book should be in all collections on gastroenterology.

Pheochromocytoma and the General Practitioner. By Joseph L. DeCourcy, M.D., and Cornelius B. DeCourcy, M.D. 8°, cloth, 165 pp. Cincinnati 2, Ohio. DeCourcy Clinic, 1952.

This monograph discusses pheochromocytoma, the "great mimic," in all its aspects. In support of its nickname 29 cases in which the disease has been mistaken for other conditions are cited. The text begins with a historical résumé, 1886-1952. There is a bibliography of 361 references and an index. The small book should be in all medical libraries.

Mechanisms of Urologic Disease. By David M. Davis, M.D., visiting lecturer in urology, Graduate School of Medicine, University of Pennsylvania. 8°, cloth, 156 pp., with 6 illustrations. Philadelphia: W. B. Saunders Company, 1953. $4.50.

The author of this small book after a long period of teaching and practicing urology, has not attempted to systematize the body of urologic knowledge. His text is organized on the basis of 8 pathologic changes, and he deals with each as it applies to the entire urogenital tract. These conditions are listed as obstruction, infection, stone formation, neoplasm, congenital malformation, trauma, foreign body and neurogenic disorders.

Shame and Guilt: A psychoanalytic and a cultural study. By Gerhart Piers, M.D., staff member, Institute for Psychoanalysis, Chicago, and Milton B. Singer, Ph.D., professor of the social sciences, College of the University of Chicago. With a foreword by Roy R. Grinker, M.D. 8°, paper, 86 pp. Springfield, Illinois: Charles C Thomas, 1953. $3.25. Publication Number 171, American Lecture Series.

This monograph constitutes the first on a psychiatric subject issued in the *American Lecture Series.* The text consists of 2 parts. The first, by Dr. Piers, is a psychoanalytic study of shame and guilt, and the second, by Dr. Singer, discusses shame and guilt cultures. Lists of references are appended to both parts.

Physical Chemistry for Students of Biology and Medicine. By David I. Hitchcock, Ph.D., associate professor of physiology, Yale University School of Medicine. Fourth edition, with laboratory experiments. 8°, cloth, 266 pp., with 31 illustrations. Boston: Little, Brown and Company, 1953. $5.00.

First published in 1932, this standard manual has been revised and brought up to date. New sections have been included in almost every chapter, and diagrams and graphs of experimental data have been added to the text. There is a good index, and the publishing is excellent.

Ciba Foundation Colloquia on Endocrinology. Vol. VI. *Hormonal Factors in Carbohydrate Metabolism.* General editor for the Ciba Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch.; assisted by Jessie S. Freeman, M.B., D.P.H. 8°, cloth, 350 pp., with 94 illustrations. Boston: Little, Brown and Company, 1953. $6.75.

This composite book is the joint work of 28 persons. The subject matter includes enzyme systems, the adrenal cortex, insulin and their influence on carbohydrate metabolism, hormonal control of the interconversion of carbohydrate, protein and fat, hormonal control of the storage of glycogen and sex hormones, pregnancy and carbohydrate metabolism. The literature is surveyed, and there are indexes of authors and subjects. The book should be in all collections on the subject.

20 Years of Psychoanalysis: A symposium in celebration of the twentieth anniversary of the Chicago Institute for Psychoanalysis. Edited by Franz Alexander, M.D., and Helen Ross. 8°, cloth, 308 pp. New York: W. W. Norton and Company, Incorporated, 1953. $3.75.

Seven physicians participated in the symposium and read papers on psychoanalysis in relation to medicine, psychiatry, the biologic sciences, social science, psychoanalytic training, and psychoanalysis as a basic science, all presented in the first part of this book. The second part consists of a history of training and research at the Institute. An appendix of 28 pages lists the publications emanating from the Institute, 1932-1952, and the faculty for 1952. The lack of an index detracts from the reference value of the book. It should be in all collections on the subject.

A Bibliography of The World Literature on Blood Pressure, 1920-1950. Edited by Ernest K. Koller and Jacob Katz, Bureau of Applied Social Research and School of Public Health, Columbia University. In 3 volumes. 4°, paper. Vol. I, *Authors and Titles.* Vol. II, *Author Index and Title Index.* Vol. III, *Abstracts.* Boston: Commonwealth of Massachusetts, 1953.

This new bibliography on an important subject is an essential reference tool for all medical libraries. The first volume lists 16,460 articles; the second is a subject-and-author index, and the third consists of approximately 1500 abstracts of selected articles. The publishing is excellent.

Holt Pediatrics. By L. Emmett Holt, Jr., professor of pediatrics, New York University College of Medicine, and director, Children's Medical Service, Bellevue Hospital, New York City; and Rustin McIntosh, Carpentier Professor of Pediatrics, Columbia University College of Physicians and Surgeons, and director of the Pediatric Service in the Babies Hospital, New York City. Twelfth edition. 8°, cloth, 1485 pp., with 269 illustrations. New York: Appleton-Century-Crofts, Incorporated, 1953. $15.00.

This treatise on pediatrics is the joint work of 72 specialists. A bibliography is appended to the various articles, which present current knowledge on the subjects. An appendix is devoted to tables on growth, blood chemistry, renal function, service areas and instructions for parents of diabetic children. There is a good index, and the book is well published. It should be in all medical libraries and in the collections of pediatricians and physicians.

Injuries of the Spinal Cord. Edited by George C. Prather, M.D., and Frank H. Mayfield, M.D. 8°, cloth, 396 pp., with illustrations. Springfield, Illinois: Charles C. Thomas, 1953. $8.75.

This monograph is the joint work of 9 physicians and surgeons. The discussion of the various aspects of spinal-cord injuries is based on a review of the literature of the different phases, including neurosurgical and urologic, nutrition, associated injuries and complications, decubitus ulcers, rehabilitation and psychologic considerations. There is a good index, and the publishing is excellent. The book is recommended for all medical libraries and neurosurgeons.

The Action of Insulin. By Niels Haugaard, Ph.D., assistant professor of physiologic chemistry in research medicine, University of Pennsylvania School of Medicine; and Julian B. Marsh, M.D., assistant professor of physiologic chemistry, Graduate School of Medicine, University of Pennsylvania. 8°, cloth, 113 pp. Springfield, Illinois: Charles C Thomas, 1953. $3.75. (Publication No. 194, *American Lecture Series.*)

The authors present the results of recent investigations into the problem of the mechanism of insulin action. The chapters deal with intermediary metabolism, chemistry of insulin, physiology of insulin action, experimental diabetes, diabetic metabolism, the relation of anterior-pituitary and adrenocortical factors to the action of insulin, the clinical aspects and mechanism of the action of insulin. The literature is thoroughly reviewed, and lists of references are appended to the chapters. There is a good index. The book should be in all collections on diabetes.

NONCLINICAL NOTES

APPOINTMENT OF DR. CASTLEMAN

Dr. Benjamin Castleman, editor of the Case Records of the Massachusetts General Hospital and a member of the editorial board of the *Journal,* has been appointed clinical professor of pathology at Harvard Medical School and chief of the Department of Pathology at Massachusetts General Hospital.

CAMBRIDGE CITY HOSPITAL HEAD

Dr. James F. Collins, former assistant superintendent of the Boston City Hospital, has been appointed medical director of the Cambridge City Hospital. The appointment becomes effective January 4.

NOTICES

EMERY MEMORIAL LECTURE

The E. Stanley Emery Memorial Lecture will be presented by Dr. Cyrus C. Sturgis, professor of medicine, University of Michigan Medical School, in the amphitheater, Peter Bent Brigham Hospital, on Wednesday, January 13, at 8 p.m. The subject will be "The Nature and Treatment of Leukemia and Allied Disorders."

NEW ENGLAND ROENTGEN RAY SOCIETY AND BOSTON ORTHOPEDIC CLUB

There will be a joint meeting of the New England Roentgen Ray Society and the Boston Orthopedic Club in Ware Hall, Boston Medical Library, 8 Fenway, Boston, on Friday, January 15, at 8 p.m.

HARTFORD MEDICAL SOCIETY

A series of lectures sponsored by the Hartford Medical Society will be held in the Hunt Memorial Building, Hartford, Connecticut, on Mondays, at 8:30 p.m. They will be preceded by a clinical conference at one of the local hospitals at 5 p.m.

January 18. The Practical Approach to Treatment of Anxiety. Dr. Joseph F. Hughes, Philadelphia.

February 1. The Surgical Treatment of Pancreatic Disease. Dr. Richard B. Cattell, Boston.

February 15. Some Problems Associated with Major Catastrophes. Dr. I. S. Ravdin, Philadelphia.

March 1. The Diagnosis and Management of Diseases of the Aseptic Meningitis Type. Dr. Theodore E. Woodward, Baltimore.

March 15. Clinical Disturbances Produced by the Overfunction and Underfunction of the Pituitary Gland. Dr. Edward H. Rynearson, Rochester, Minnesota.

April 5. Angiography. Dr. Israel Steinberg, New York City.

April 19. Indications for Surgery in Valvular Heart Disease: With specific reference to mitral stenosis. Dr. Edward F. Bland, Boston.

MASSACHUSETTS HOSPITAL ASSOCIATION

The annual meeting of the Massachusetts Hospital Association will be held at the Hotel Statler, Boston, on Tuesday, January 26. The morning sessions will include panels on emergency planning, improvement of patient care and the problems of hospital reimbursement. The afternoon speakers will be Governor Herter of Massachusetts and Dr. Robert Page, professor of industrial medicine, New York University, and medical director of Standard Oil Company. The dinner speaker will be Dr. Abram L. Sachar, president of Brandeis University.

SOCIETY MEETINGS AND CONFERENCES

JANUARY 6, MARCH 3 and APRIL 21. Massachusetts Society for Research in Psychiatry. Page 750, issue of October 29.

JANUARY 12. Harvard Medical Society. Page 1087, issue of December 24.

JANUARY 13. Boston Gastroenterological Society. Page 1044, issue of December 17.

JANUARY 13. E. Stanley Emery Memorial Lecture. Notice above.

JANUARY 15. New England Roentgen Ray Society and Boston Orthopedic Club. Notice above.

JANUARY 18, FEBRUARY 1 and 15, MARCH 1 and 15 and APRIL 5 and 19. Hartford Medical Society. Notice above.

JANUARY 19. South End Medical Club. Page 1088, issue of December 24.

JANUARY 22, 23, 29 and 30. International College of Surgeons Regional Meeting. Page 828, issue of November 12.

JANUARY 26. Massachusetts Hospital Association. Notice above.

FEBRUARY 2-4. Examination for Public Health Service Medical Officers. Page 750, issue of October 29.

FEBRUARY 5 and APRIL 9. New England Society of Anesthesiologists. Page 914, issue of November 26.

FEBRUARY 15-20. Pan-American Academy of General Practice. Page 790, issue of November 5.

FEBRUARY 23-25. College of Medical Evangelists Alumni Postgraduate Convention. Page 1044, issue of December 17.

MARCH 15-26. Course in Laboratory Diagnosis of Virus Diseases. Page 1044, issue of December 17.

MARCH 27 and 28. American Psychosomatic Society. Page 1044, issue of December 17.

MAY 14-16. Fifth Annual International Group of Doctors in Alcoholics Anonymous. Page 1004, issue of December 10.

MAY 31-JUNE 3. American Urological Association. Page xvii, issue of July 23.

JULY 22 and 23. International College of Surgeons Regional Meeting. Page 828, issue of November 12.

JULY 23-29. International Cancer Congress. Page 914, issue of November 26.

SEPTEMBER 7-10. Nineteenth Annual Congress of the United States and Canadian Sections of the International College of Surgeons. Page 828, issue of November 12.

SEPTEMBER 12-15. Second International Congress of Cardiology. Page 44, issue of July 2.

SEPTEMBER 16-18. American Heart Association Annual Scientific Sessions. Page 44, issue of July 2.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH

JANUARY 13. Pentucket Club, 111 Main Street, Haverhill.

MAY 12. 113th Annual Meeting. Andover Country Club, Andover.

MIDDLESEX EAST

JANUARY 20. Combined with the Auxiliary.

MARCH 24.

MAY 11. Annual Meeting. (Harlow Dinner.)

MIDDLESEX SOUTH

JANUARY 27. Jenkins Auditorium, Malden High School, Salem Street, Malden. 8:00 p.m.

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, JANUARY 7

THURSDAY, JANUARY 7

*8:00-8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*9:00-10:30 a.m. Cardiac Grand Rounds. Main Amphitheater Children's Medical Center.

*9:00-10:00 a.m. Arthritis Grand Rounds. Robert Breck Brigham Hospital.

10:30-11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*11:00 a.m.-12:30 p.m. Hand Clinic (Physical Medicine and Rehabilitation Service). Boston City Hospital.

*5:00-6:00 p.m. Surgical Division Meeting. Mount Auburn Hospital, Cambridge.

*8:15 p.m. Deaconess Medical Lecture. Thyroid Disease. Dr. Lewis Hurxthal. Joslin Auditorium, New England Deaconess Hospital.

FRIDAY, JANUARY 8

*8:00-8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*9:30-11:30 a.m. Medical and Surgical Grand Rounds. Drs. George W. Thorn and Francis D. Moore. Main Amphitheater, Peter Bent Brigham Hospital.

*10:00 a.m. Tuberculosis Surgical Clinic. South End Health Unit, 57 East Concord Street.

10:30-11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

12:00 m.-1:00 p.m. Clinicopathological Conference (Boston Floating Hospital). Joseph H. Pratt Diagnostic Hospital.

*12:30-1:30 p.m. Surgery of Upper Genitourinary Tract. Dr. R. Olson. Ward B Classroom, Cambridge City Hospital.

*1:30 p.m. Fertility and Endocrine Clinic. Free Hospital for Women, Brookline.

*1:30 p.m. Tumor Clinic. Mount Auburn Hospital, Cambridge.

*3:00-4:00 p.m. X-ray Conference. Dr. F. Herzan. Ward B Classroom, Cambridge City Hospital.

*4:00-10:00 p.m. Alcoholism Clinic. By appointment. Washingtonian Hospital, 41 Waltham Street.

SATURDAY, JANUARY 9

*8:00-8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*8:30-10:00 a.m. Orthopedic Staff Conference. Boston City Hospital.

*9:00 a.m. Proctology Clinic. Peter Bent Brigham Hospital.

*9:00 a.m. Alcoholism Clinic. Peter Bent Brigham Hospital.

10:30-11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

11:00 a.m.-12:00 m. Blood Clinic. Dr. William Dameshek. Stearns Auditorium, New England Center Hospital.

SUNDAY, JANUARY 10

Your Visit to the Doctor. Nichols Sun Room. Faulkner Hospital, 1153 Centre Street, Jamaica Plain.

(Notices concluded on page ix)

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NOTICES (*Concluded from page 1130*)

MONDAY, JANUARY 11

*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*8:00–9:00 a.m. Medical and Surgical Clinic on Diabetic Foot Problems. New England Deaconess Hospital.

8:30–9:30 a.m. Clinic by entire Surgical Staff and Anesthesia Staff. Cheever Amphitheater, Dowling Building, Boston City Hospital.

10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.

*4:30 p.m. Seminar. Department of Biological Chemistry of the Harvard Medical School. Nucleic Acids: Their Content and Composition in Liver Cell Fractions. Dr. C. A. Villee and Mr. P. Olmsted. Bowditch Library, Harvard Medical School, 25 Shattuck Street.

TUESDAY, JANUARY 12

*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*9:00 a.m. Geriatrics Clinic. Peter Bent Brigham Hospital.

10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*11:30 a.m.–12:30 p.m. Clinical Pathological Conference. Dr. C. Jones. Nurses' Home, Cambridge City Hospital.

*12:00 m. Pediatric Grand Rounds. Cheever Amphitheater, Dowling Building, Boston City Hospital.

*12:00 m.–1:00 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.

*12:15–1:15 p.m. X-ray Conference. Dr. Merrill Sosman. Main Amphitheater, Peter Bent Brigham Hospital.

*12:30–1:30 p.m. Medical Journal Club. Cafeteria, New England Deaconess Hospital.

*4:30–5:30 p.m. Anesthesia Teaching Conference. Stearns Auditorium, New England Center Hospital.

*5:00 p.m. Surgical Journal Review. Mount Auburn Hospital, Cambridge.

*5:00–6:00 p.m. Service Meeting followed by Clinical Conference (Medical, Surgical, Obstetrical). Faulkner Hospital.

8:00 p.m. Harvard Medical Society. Main Amphitheater, Peter Bent Brigham Hospital.

WEDNESDAY, JANUARY 13

*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*12:00 m. Boston State Hospital Psychiatric Seminar. Reception Building Auditorium, 591 Morton Street, Dorchester.

*12:00 m.–1:00 p.m. Medical Conference. Jimmy Fund Building, Children's Medical Center. 35 Binney Street.

*12:30 p.m. Weekly Pathological Meeting. Joslin Auditorium, New England Deaconess Hospital.

12:30–1:30 p.m. Weekly Staff Review of Autopsies. Dr. William A. Meissner. Joslin Auditorium, New England Deaconess Hospital.

*1:30–2:30 p.m. Pathological Conference. Dr. C. Jones. Nurses' Home, Cambridge City Hospital.

2:00–3:00 p.m. Pediatric Conference for Practitioners. Jimmy Fund Building, Children's Medical Center. 35 Binney Street.

*4:00–5:30 p.m. Fracture Lecture. Boston City Hospital.

*4:00–6:00 p.m. Overholt Thoracic Conference. Joslin Auditorium, New England Deaconess Hospital.

*5:00–6:00 p.m. American Cancer Society Telecast. Sarcoma of Soft Parts. Dr. Arthur Purdy Stout. Children's Cancer Research Center, 35 Binney Street.

8:00 p.m. Boston Gastroenterological Society. Gastrointestinal Bleeding. Drs. Thomas Warthen and J. Englebert Dunphy. Cheever Amphitheater, Boston City Hospital.

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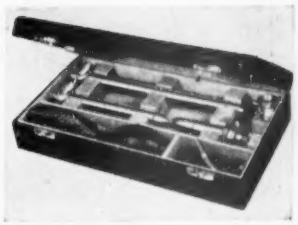

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